"Evaluation of Mannheim Peritonitis Index (MPI) in predicting early management and outcome in patients with peritonitis"

Dissertation submitted in Partial fulfilment of the regulations

required for the award of

M.S. DEGREE

In

General Surgery Branch - I

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL, 2014

CERTIFICATE

This is to certify that this dissertation titled **"Evaluation of Mannheim Peritonitis Index (MPI) in predicting early management and outcome in patients with peritonitis"** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch - I (General Surgery) is a bonafide work done by Dr.RANJITH CHERIYAN PHILIP, post graduate student in General Surgery under my direct supervision and guidance during the period of November 2012 to November 2013.

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DECLARATION

I hereby declare that the dissertation entitled **"Evaluation of Mannheim Peritonitis Index (M P I) in predicting early management and outcome in patients with peritonitis** "was done by me at Coimbatore Medical College Hospital Coimbatore – 641018 during the period of my post graduate study for M.S. Degree Branch-1 (General Surgery) from November 2012 to November 2013.

This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the University regulations for award of M.S., Degree in General Surgery.

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LIST OF ABBREVATIONS USED

MPI	Mannheim Peritonitis Index
USG	Ultrasound
CT	Computerized tomography
ICU	Intensive care unit
APACHE(II)	Acute Physiological and Chronic Health Evaluation score
POSSUM	Physiological and Operative Severity Score for enumeration of Mortality and morbidity

ABSTRACT

BACKGROUND AND OBJECTIVE:

Peritonitis is one of the common cause of ‘acute abdomen’. It is one of themajor cause of morbidity and mortality worldwide.. Despite all the recent advances in the diagnostic and management techniques, peritonitis is a great challenge to medical fraternity.An accurate predictive ability would make it possible to measure more precisely, the quality of intensive care and other new life-saving technologies.Scoring systems that group patients based on the severity of illness before treatment can allow a meaningful analysis of morbidity and mortality rates. Hence I selected a study to evaluate Mannheim Peritonitis Index which is severity scoring system, in predicting outcome in patients with peritonitis.

METHODS

Patients with secondary peritonitis managed in the surgical wards and ICU in Coimbatore Medical College Hospital are included in my study. Resuscitation measures, antibiotic therapy, vasoactive drugs, nasogastric intubation and analgesics administered as required. MPI were calculated at admission or during management. All patients will undergo laparotomy and managed according to the cause. After surgery interventions like antibiotic therapy, vasoactive drugs, resuscitation and ICU care given as necessary.

Patients followed up until discharge or death. Patients are grouped into three categories based on disease severity those with MPI<21, between 21 and 29 ,> 29. Mortality rates calculated belonging to each group.

RESULTS

This study included 100 patients with peritonitis, which showed a male predominance. The ratio is approximately 2:1. Most of the patients belong to age group between 15 to 30.Major cause of post-operative morbidity was wound infection and respiratory complications. 62 cases were in low risk group with nil mortality, 20 cases in moderate risk group with 45% mortality and 18 patients in high risk group with 89% mortality. Duration of hospital stay doesn't correlate with severity of disease because a patient with MPI score more than 29 succumbs to death in immediate postoperative period. The complications have been most common in the group of patients having a MPI score between 22 to 29, whereas those who have a score above 29 have higher mortality.

CONCLUSION

I conclude that Mannheim Peritonitis index (MPI) is simple and objective scoring system to predict the final outcome of patients with peritonitis and intraabdominal sepsis. It appears more practical than other scoring systems.MPI provides an easy and reliable means of risk evaluation and classification for

patients with peritoneal inflammation for early intensive management for better outcome of patient.

KEY WORDS:

Mannheim Peritonitis Index, peritonitis, severity, mortality

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INTRODUCTION

Definition:

Peritonitis is defined as inflammation of peritoneum and peritoneal cavity, usually caused by a localized or generalized infection.

Peritonitis is one of the common cause of 'acute abdomen'. It is one of themajor cause of morbidity and mortality worldwide. The disease is perhaps asancient as mankind. With newer methods in diagnosis such as the sophisticated radiological investigations and progress in treatment strategies such as newer and more effective antibiotics, fluid management, and parenteral nutrition have brought down complication rates. Despite all the recent advances in the diagnostic and management techniques, peritonitis is a great challenge to medical fraternity. An accurate predictive ability would make it possible to measure more precisely, the quality of intensive care and other new life-saving technologies. Predictable Risk stratification andprecise prognosis before treatment would also enable clinical researchers to use observational studies to compare the quality of care in various intensive care units (ICUs) and to identify those components of ICU structure that are linked to improved patient outcome. Such informations help in early aggressive management and improved the outcome of patient.

To predictseverity of the disease several scoring systems are developed. Scoring systems that group patients based on the severity of illness before treatment can allow a meaningful analysis of morbidity and mortality rates.The etiology of peritonitis varies from western region to that of India. There is a lack of data from India regarding prognostic indicators, and mortality and morbidity patterns. Most of the patients with peritonitis in peripheral hospitals there is lack of advanced investigative modalities. So there is a need of scoring method to predict morbidity and mortality and also to decide about the treatment with minimal investigative modalities.

With this we intend to study the efficacy of Mannheim Peritonitis Index (MPI) in predicting the morbidityand mortalityin patients with peritonitis.

AIMS AND OBJECTIVES

- Early classification of severity of peritonitis by Mannheim peritonitis index(MPI) scoring system
- Selecting patients for aggressive surgical approach based on MPI score
- To predict outcome of patients with peritonitis

REVIEW OF LITERATURE

HISTORY OF PERITONITIS [15]

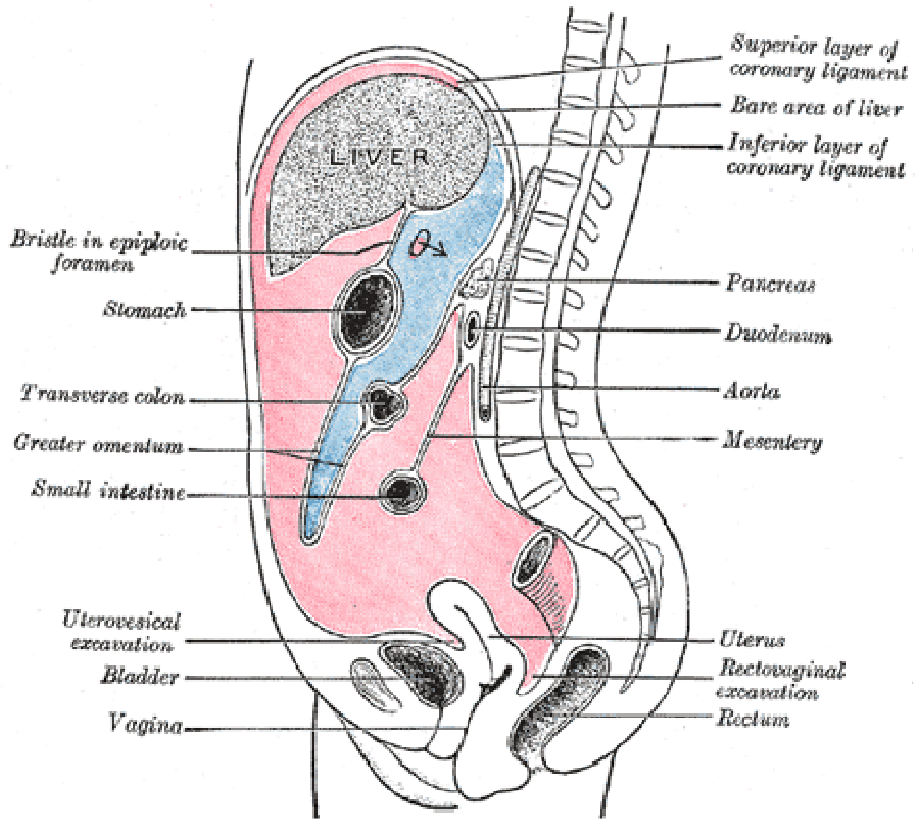
The surgical treatment of peritonitis started with the first laparotomy by McDowell was for infected ovarian cyst in the beginning of the19th century. After that the surgical treatment modalitiesdeveloped parallel to the advances in abdominal surgery. In the last decade of the 19thcentury Mikulicz felt that laparotomy was indicated in all patients with purulent peritonitis. In the beginning of the 20thcentury Körte and Kirschner defined the principles of surgery for peritonitis that are valid up to this day: early surgical intervention, elimination of the infectious sources, and peritoneal lavage. Surgeons have discussed the utility of draining and irrigating the peritoneal cavity from since that time. Postoperative peritoneal lavage was already advocated in the beginning of the last century, but generally considered ineffective. Thirty years ago postoperative lavage was again strongly advocated, but evidence for its benefit is still lacking.

EMBRYOLOGY [1, 2]

The embryogenesis of the peritoneum derives from the mesoderm. Around the third week, the mesoderm differentiates into lateral plate mesoderm, intermediate mesoderm, and paraxial mesoderm. As differentiation proceeds, division of lateral plate occur into somatic and splanchnic mesoderm. These envelop the intraembryonic coelom on each side of the midline. Later, the right and left intraembryonic coelom will unite to form a single cavity. This single cavity will subdivide again into pleural, pericardial, and peritoneal cavities and into the processus (tunica) vaginalis. Differentiation continues by the formation of parietal and visceral layers of the serous membranes of the peritoneum, and by the formation of omenta, mesenteries, ligaments, and fossae.

ANATOMY [1, 2, 7]

The peritoneum consists of a single sheet of simple squamous epithelium of mesodermal origin, termed mesothelium, lying on a thin connective tissue stroma .The surface area is 1.0 to 1.7 m^2, about that of the total body surface area. In males, the peritoneal cavity is sealed, whereas in females, it is open to the exterior through the ostia of the fallopian tubes.



Peritoneal cavity female

The peritoneal membrane is divided into parietal and visceral components. The parietal peritoneum covers the anterior, lateral, and posterior abdominal wall surfaces as well as the inferior surface of the diaphragm and the pelvis.

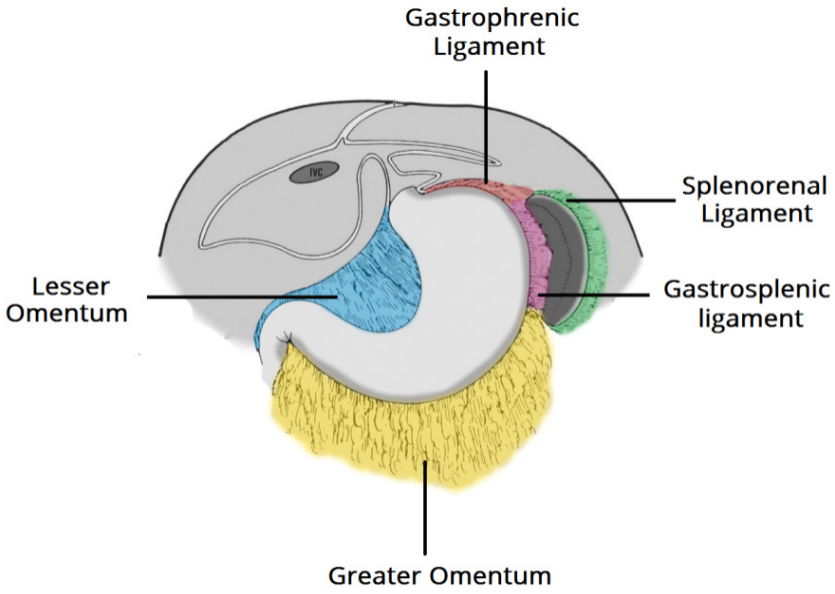
The visceral peritoneum covers most of the surface of the intraperitoneal organs (i.e., the stomach, jejunum, ileum, transverse colon, liver, and spleen) and the anterior aspect of the retroperitoneal organs (i.e., the duodenum, left and right colon, pancreas, kidneys, and adrenal glands).

The peritoneal cavity is subdivided into interconnected compartments or spaces by 11 ligaments and mesenteries.

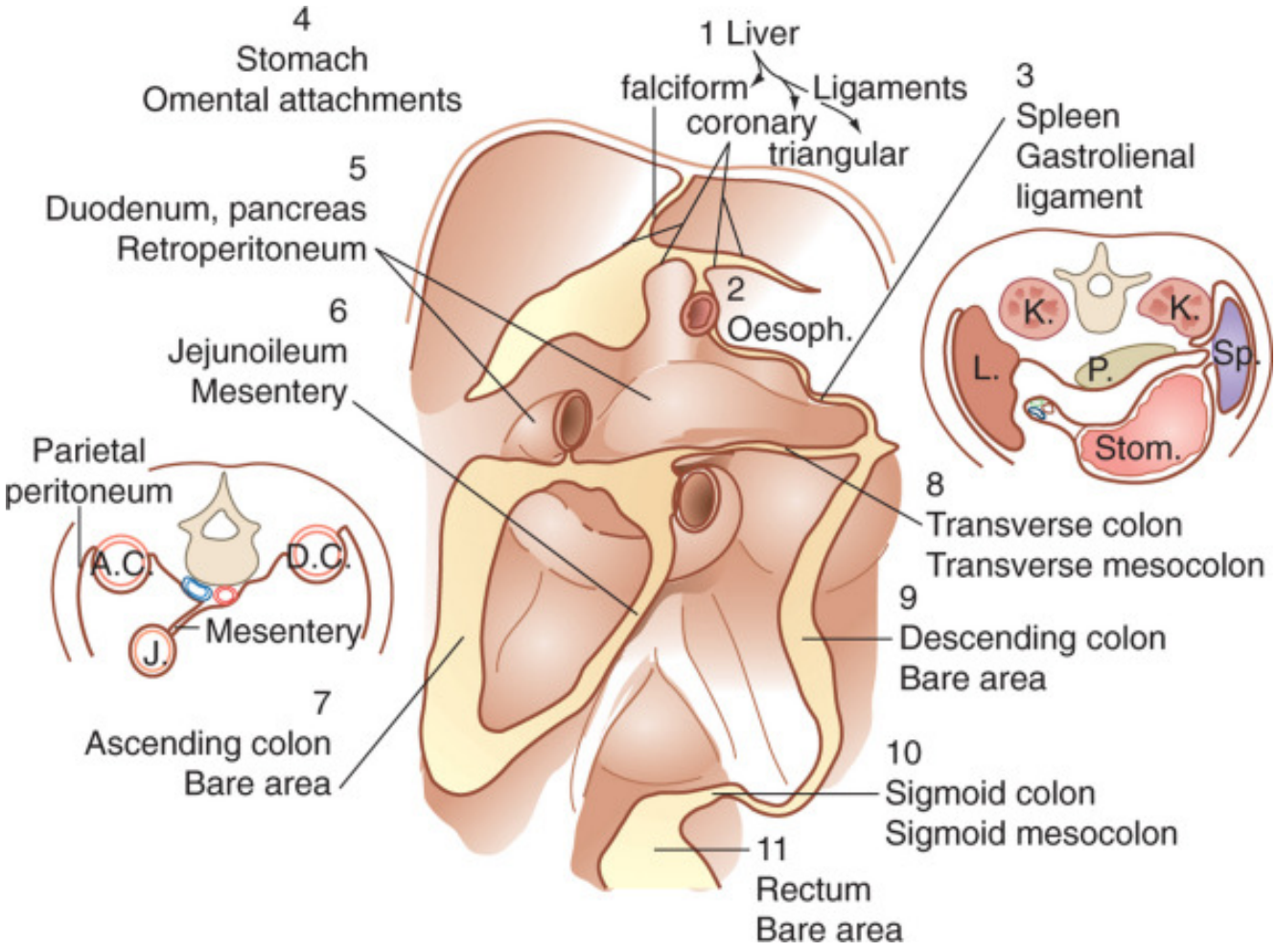
The peritoneal ligaments or mesenteries include the coronary, gastrohepatic, hepatoduodenal, falciform, gastrocolic, duodenocolic, gastrosplenic, splenorenal, and phrenicocolic ligaments and the transverse mesocolon and small bowel mesentery .

These structures partition the abdomen into nine potential spaces: right and left subphrenic, subhepatic, supramesenteric and inframesenteric, right and left paracolic gutters, pelvis, and lesser space.

These ligaments, mesenteries, and peritoneal spaces direct the circulation of fluid in the peritoneal cavity and thus may be useful in predicting the route of spread of infectious and malignant diseases.



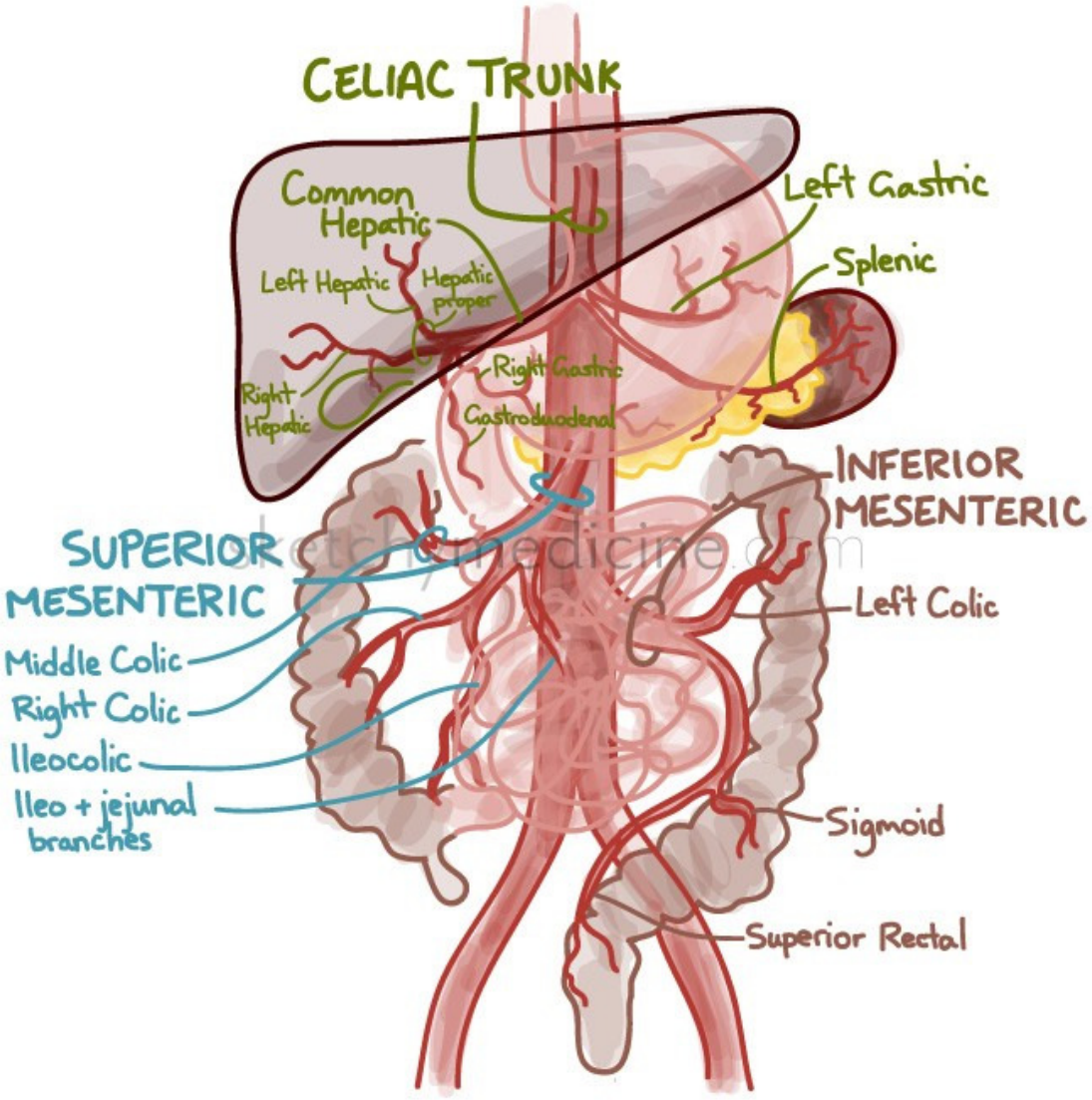
Peritoneal ligaments



Peritoneal Ligaments

BLOOD SUPPLY[1,2]

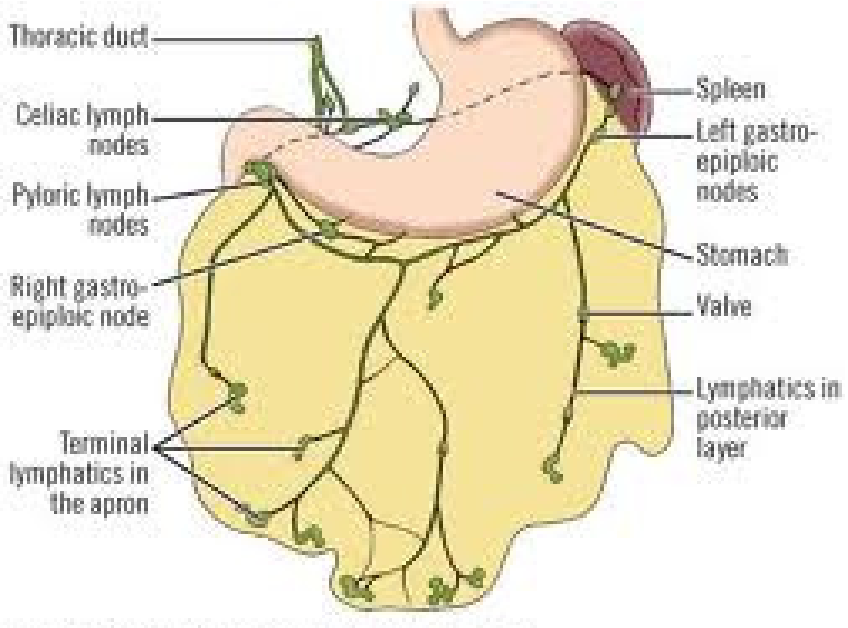
The blood supply to the abdominal parietal peritoneum is from the branches of the arteries of the abdominal wall. The blood supply of the pelvic parietal peritoneum is from the blood vessels of the pelvic wall. Blood to the visceral peritoneum is from branches of the celiac trunk and from branches of the superior and inferior mesenteric arteries, or the pelvic visceral blood vessels.



Blood supply of viscera and peritoneum

LYMPHATIC DRAINAGE

The lymphatics of the parietal peritoneum join the lymphatics of the body wall, and all drain to parietal lymph nodes. However, the lymphatics of the visceral peritoneum join the lymphatics of the related organs and are drained accordingly.

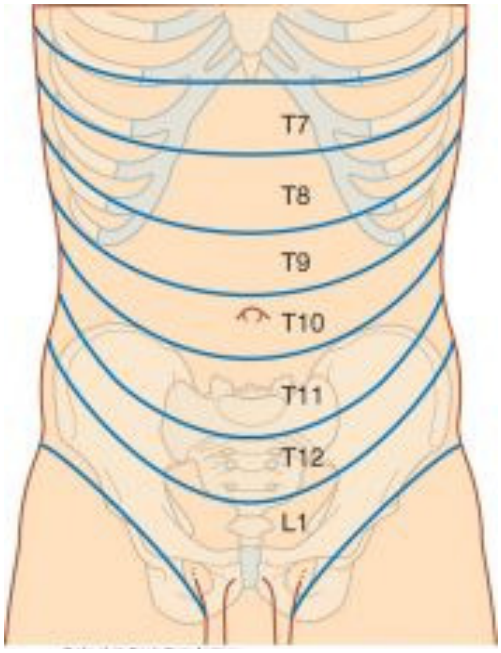


Lymphatic drainage of peritoneum

NERVE SUPPLY

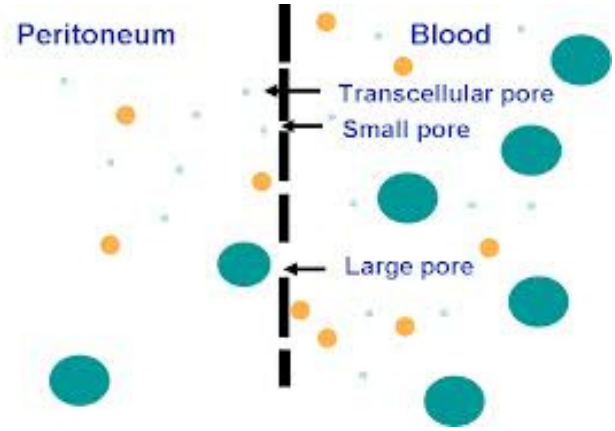
The parietal peritoneum contains somatic afferent nerves. The peritoneum contains many sensory fibers for the sensation of pain; the anterior portion of the parietal peritoneum is especially sensitive. The parietal peritoneum, therefore, is similar in sensitivity to the parietal pleura of the thorax.

In contrast, the visceral peritoneum has no somatic afferent nerves and is relatively insensitive to pain. Sensations which do occur are poorly perceived and not clearly localized by the brain, as is characteristic of visceral afferent fibers carried by autonomic nerves to viscera in general. The principal stimulus which can evoke pain from visceral peritoneum is tension upon or stretching of the tissue, or ischemia. A perforated viscus may, perhaps, produce anterior abdominal wall rigidity, and an intraperitoneal fluid collection may produce painlike sensations of traction or tension on the mesentery in the retroperitoneal space, but not localized pain.



Nerve supply of corresponding anterior abdominal wall

Peritoneum is a semipermeable membrane which selectively circulates materials between peritoneal cavity and blood.



Physiology of peritoneum

Normal volume of peritoneal fluid is about 50 ml of which is a transudate with following characteristics.

- Specific gravity below 0.016;
- protein concentration below 3g/dl;
- white blood cell count below 3000/µL;
- complement mediated antibacterial activity; and
- lack of fibrinogen related clotting.

Peritoneal fluid is circulated towards the sub diaphragmatic lymphatics.The circulation of fluid in the peritoneal cavity is driven inpart by the movement of the diaphragm. Intercellular pores inthe peritoneums covering the inferior surface of the diaphragm (termed stomata) communicate with lymphatic pools in thediaphragm. Lymph flows from these diaphragmatic lymphaticchannels through subpleural lymphatics to the regional lymphnodes and ultimately the thoracic duct. Relaxation of the diaphragm during exhalation opens the stomata and the negativeintrathoracic pressure draws fluid and particles, includingbacteria, into the stomata. Contraction of the diaphragm duringinhalation propels the lymph through the mediastinal lymphaticchannels into the thoracic duct.

It is postulated that this so-calleddiaphragmatic pump drives the movement of peritoneal fluid ina cephalad direction toward the diaphragm and into the thoraciclymphatic vessels. This circulatory pattern of peritoneal fluidtoward the diaphragm and into the central lymphatic channelsis consistent with the rapid appearance of sepsis in patients withgeneralized intra-abdominal infections, as well as the perihepatitisof Fitz-Hugh–Curtis syndrome in patients with acute salpingitis.

Peritoneal cavity is normally sterile.

The peritoneum and peritoneal cavity respond to infection in five ways:

1. Bacteria are rapidly removed from the peritonealcavity through the diaphragmatic stomata andlymphatics.

2. Peritoneal macrophages release proinflammatorymediators that promote the migration of leukocytesinto the peritoneal cavity from the surrounding microvasculature.

3. Degranulation of peritoneal mast cells releases histamine and other vasoactive products, causing localvasodilation and the extravasation of protein-richfluid containing complement and immunoglobulinsinto the peritoneal space.

4. Protein in the peritoneal fluid opsonizes bacteria, which, along with activation of the complement cascade, promotes neutrophil- and macrophagemediatedbacterial phagocytosis and destruction.

5. Bacteria become sequestered within fibrin matrices, thereby promoting abscess formation and limitingthe generalized spread of the infection.

Peritonitis occurs if peritoneal defense mechanisms are overcome by massive or continuous contamination.

Bacterial contamination causes release of many bacterial isliposaccharides. These cause increased expression of tumour necrosis factor (TNF).

Increased TNF causes increased expression of plasminogen activator inhibitor, thus resulting in decreased plasminogen and persistence of fibrin.

Fibrin clots segregate bacterial deposits, thus reducing the source of endotoxins that contribute to sepsis, but this may inadvertently shield the bacteria from the body defence mechanisms.

Role of Omentum is evident as 'Policeman of abdomen'.

It helps in

- sealing off a leaking viscus (eg, a perforated ulcer) or an area of inflammation (eg, appendicitis)
- giving collateral blood supply to ischemic viscera.
- bacterial scavenging function by absorption of infective particles
- delivery of phagocytes that scavenge bacteria.

ACUTE SECONDARY BACTERIAL PERITONITIS[2,4]

Pathophysiology

Peritonitis is an inflammatory response of peritoneal lining due to direct irritation.Secondary peritonitis occurs due to bacterial contamination originating from within the viscera or from external sources (eg, penetrating injuries). It most often follows disruption of hollow viscus.

The extravasated fluids are often sterile but will provoke a severe inflammatory response once they get infected after bacterial migration.Gastric juice from a perforated duodenal ulcer remains mostly sterile for several hours,

during which time it produces a chemical peritonitis with large fluid losses; but

If left untreated it evolves within 6-12 hours into bacterial peritonitis. Intraperitonial fluid dilutes opsonic proteins and impairs phagocytosis.

When hemoglobin gets collected in peritoneal cavity, Escherichia coli growing within the cavity can elaborate toxins that reduce bactericidal activity. Continued contamination leads to generalized peritonitis and eventually to septicemia and multi organ failure.

Commonest causes of peritonitis given in table 1

Table No 1

Causes	Mortality rate
Appendicitis	<10%
gastroduodenal ulcers perforation	
Acute salpingitis	
Diverticulitis	<20%
small bowel perforation	
Gangrenous cholecystitis	
Multiple trauma	
Perforated Large bowel	20-80%
Ischemic bowel disease	
Acute necrotizing pancreatitis	
Postoperative complications	

Factors influencing the severity of peritonitis include the

- extent of contamination,
- duration of injury,
- presence of organ failure
- host factors.

Causative organisms

Systemic sepsis in peritonitis depends on the virulence of the causative organism and the bacterial load, and the duration of bacterial proliferation and synergistic interaction. Most peritonitis is caused by poly microbial infection. Cultures usually contain mixture of aerobic and anaerobic organisms. This usually mimics the microbial contents of the organ involved. Proximal bowel perforations usually showing gram positive organisms and as it goes to distal bowel there will be more of gram negative and anaerobic organisms. Predominant aerobic pathogens include gram negative bacteria E.coli, proteus, and the Enterobacter-Klebsiella groups. The anaerobic group is dominated by Bacteroidesfragilis, anaerobic cocci, and clostridia. Any synergisms between anaerobic and anaerobic organisms increase the severity of the peritonitis.

Tuberculous peritonitis [2]

Tuberculosis is common in impoverished areas of the world. It isincreasing frequency in the United States and other developed countries.Peritoneal tuberculosis is the sixth most common site of extra pulmonary tuberculosis after lymphatic, genitourinary, bone and joint, miliary, and meningeal. Most cases of Tuberculous peritonitis result from reactivation of latent peritoneal disease that had been previously established hematogenously from a primary pulmonary focus. About one-sixth of cases are associated with active pulmonary disease.

The illness often presents insidiously, with patients having had symptoms for several weeks to months at the time of presentation. Abdominal swelling due to ascites formation is the most common symptom, occurring in more than 80% of instances.Similarly, most patients complain of a non-localized, vague abdominal pain. Constitutional symptoms such as low-grade fever and night sweats, weight loss, anorexia, and malaise are reported in about 60% of patients.Abdominal tenderness is present upon palpation in about half of patients with peritoneal tuberculosis.A positive tuberculin skin test is present in most cases, whereas only about half of these patients will have an abnormal chest radiograph.

The ascitic fluid SAAG is less than 1.1 g/dL, consistent with a high protein concentration within the ascitic fluid. Microscopic examination of the ascites shows erythrocytes and an increased number of leukocytes, most of which are lymphocytes.

Abdominal imaging with ultrasound or CT may suggest the diagnosis but lacks the sensitivity and specificity to be diagnostic. Ultrasound may demonstrate the presence of echogenic material within the ascitic fluid, seen as fine mobile strands or particulate matter. CT will demonstrate the thickened and nodular mesentery with mesenteric lymphadenopathy and omental thickening.

The diagnosis is made by laparoscopy with directed biopsy of the peritoneum. In more than 90% of cases, laparoscopy demonstrates multiple whitish nodules (<5 mm) scattered over the visceral and parietal peritoneum; histologic examination of these nodules demonstrates caseating granulomas

Treatment of peritoneal tuberculosis includes antituberculous drugs. Drug regimens useful in treating pulmonary tuberculosis are also effective for peritoneal disease, with Isoniazid and Rifampin daily for 9 months being a commonly used and effective regimen.

A case of Tuberculous peritonitis showing peritoneal tubercles

Clinical findings[4, 6, 9]

Clinical and laboratory evaluation needed for estimating the severity of the peritonitis which helps in specific treatment and surgery can be determined.

Clinical features reflect the duration and severity of peritonitis. Age and general health of the patient bear considerably on the outcome of the disease.

Usual presentation is like an acute abdomen.

Local findings include

1. abdominal pain,
2. tenderness,
3. guarding and rigidity,
4. distension,

5. free air in abdomen,
6. free fluid in abdomen
7. Diminished bowel sounds.

Systemic findings include

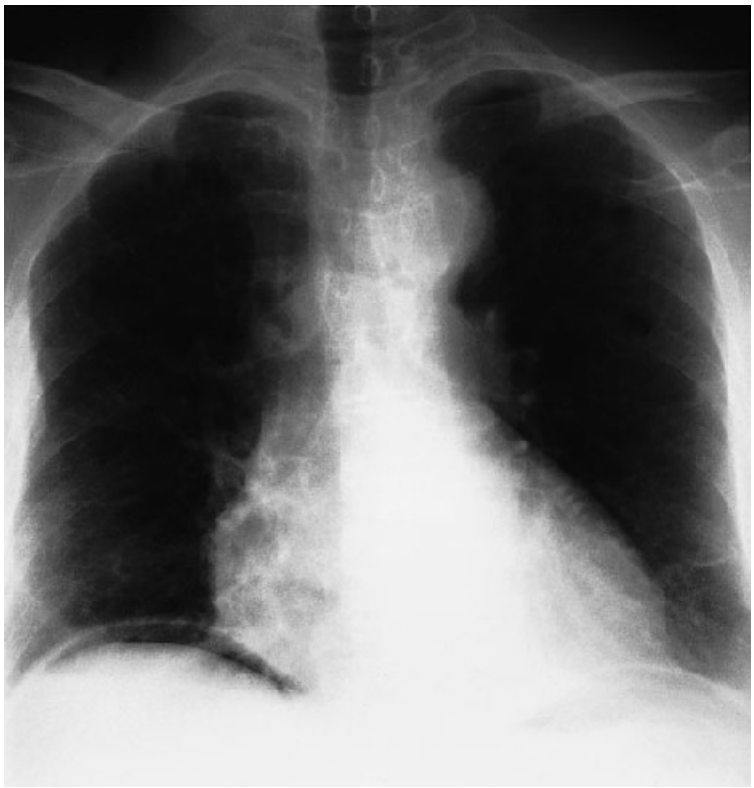
1. fever
2. chills or rigors
3. tachycardia
4. tachypnea
5. restlessness
6. dehydration
7. oliguria
8. disorientation
9. refractive shock

Shock is due to combined effect of hypovolemia and septicemia with multi organ dysfunction.

Figure showing Hippocratic facies

These signs are difficult to interpret in very young and very old as well as those who are chronically debilitated or immunosuppressed.

Radiological investigations like x-ray, ultra sonogram, CT scan can show free air below diaphragm or free fluid in pelvic cavity and Morrison's pouch, it can also show dilated bowel loops and absent peristalsis, it can give the organ involved in the pathology.(eg, appendicitis, diverticulitis)



X-ray showing Air under diaphragm

X-ray showing multiple air fluid level with air under diaphragm

Laboratory findings asses the severity of peritonitis and guide therapy. Blood studies should include a complete blood cell count, arterial blood gas, electrolytes, liver and renal function tests. Samples for culture for blood, urine, sputum, and peritoneal fluid should be taken before starting of antibiotics.

Differential Diagnosis

Specific types of infective peritonitis can be seen (eg, gonococci, candida) and non-infective peritonitis can also be there. In elderly systemic diseases (eg, pneumonia, uremia) can produce paralytic ileus so striking that it may resemble peritonitis or bowel obstruction.

Treatment of Peritonitis

Fluid and electrolyte replacement, operative control of sepsis, and systemic antibiotics are the mainstay of treatment of peritonitis.

Pre-Operative Care

Intravenous fluids:

The massive transfer of fluids into the peritoneal cavity should be replaced by an appropriate amount and type of intravenous fluid. In patients with systemic toxicity or if the patient is old or in fragile health, a central venous line should be started for the dual purpose of monitoring the central venous pressure as well as infusion of adequate amount of fluids. A bladder catheter introduced for monitoring the urine output, and serial body weight measurements are done to monitor fluid requirements. Ringer lactate or balanced solution is infused rapidly to correct intravascular hypovolemia and to maintainurine output. Blood may be required in patients who are anemic or in those who have associated bleeding.

In advanced septicemia inotropics and mechanical ventilation may be necessary and should be provided in an intensive care setup.

Antibiotics:

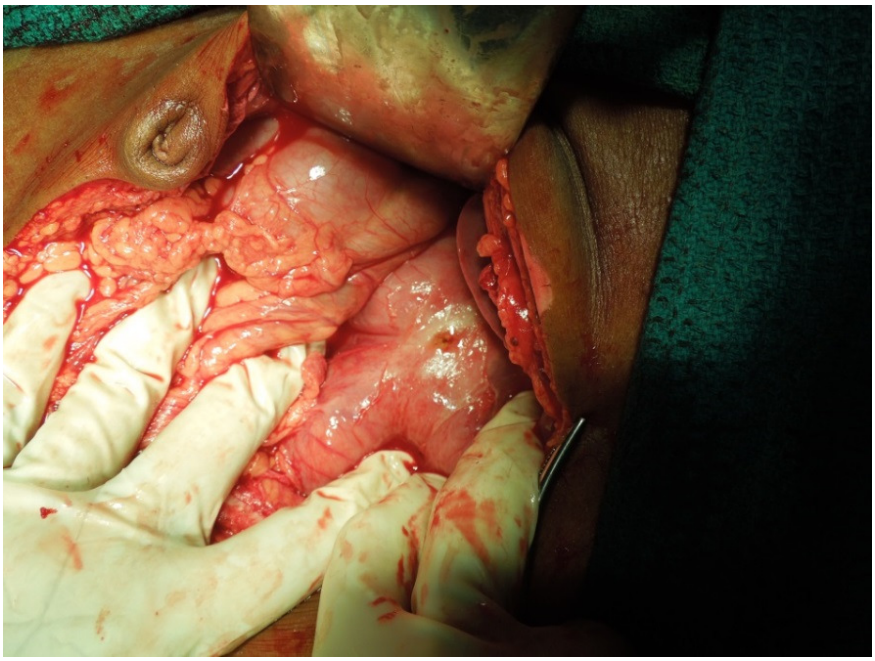
Loading doses of intravenous antibiotics should be given directed against the anticipated pathogen after the samples for culture and sensitivity are taken. Initial antibiotics usually used are third generation cephalosporin, ampicillin-sulbactam, ticarcillin-clavulinic acid, aztreonam or imipenem-cilastatin for gram negative coliforms and metronidazole or clindamycin for anaerobic organisms. Inadequate drug dosing in the initial period may contribute for treatment failure. Aminoglycosides should be used with care because of the fear for renal side effects associated with their use. Antibiotics should be modified postoperatively according to culture and sensitivity patterns. Antibiotics are continued till the patient is afebrile and a differential count of less than 3% bands is achieved.

Operative Management [5, 10.13]

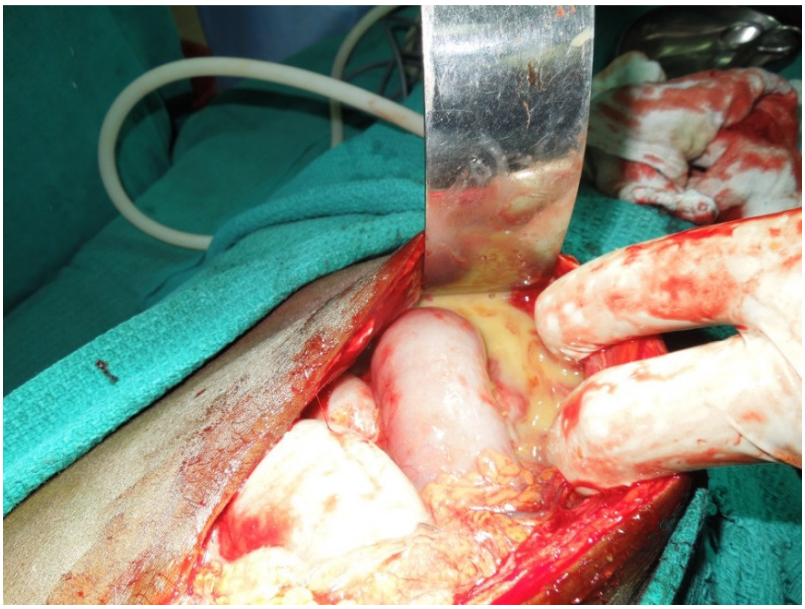
Control of sepsis:

The surgery should be aimed at removing all the infective material, correct the underlying cause, and prevent late complications. In generalizedperitonitis midline incision offers the best surgical exposure. A thorough laparotomy is performed and all the necrotic and infective materials should be removed. Special attention should be given to peritoneal recesses where there is a chance of localized infections. Adequate samples for cultures are taken and sent for

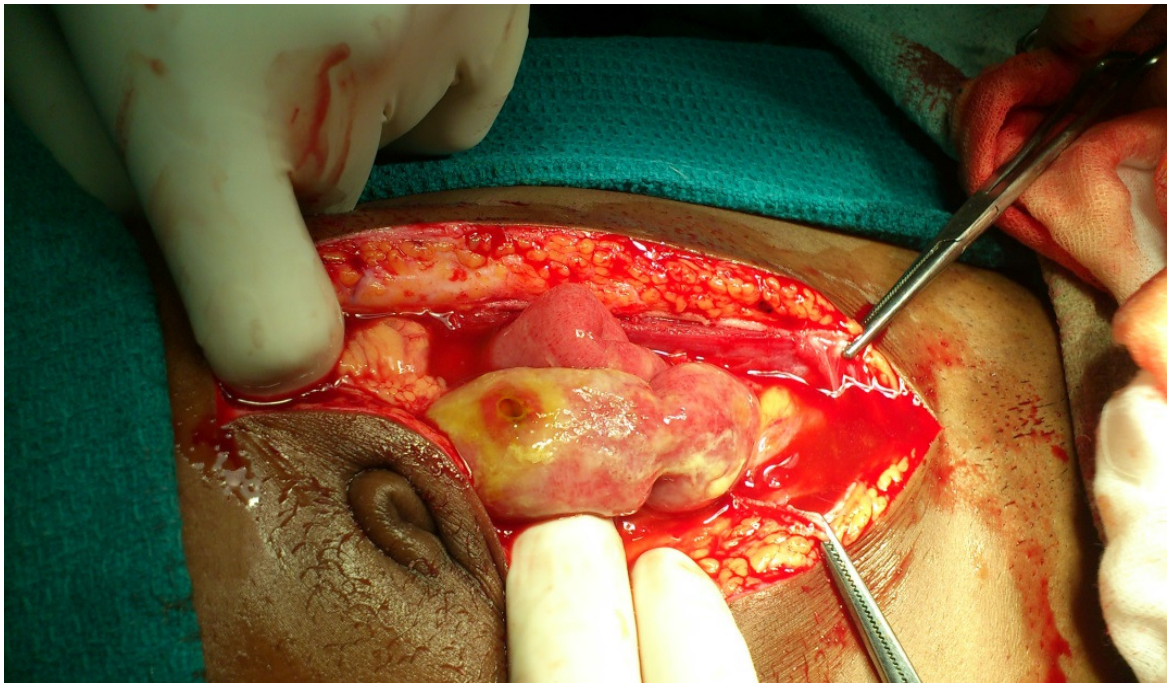
sensitivity tests. In case of localized peritonitis incision can be directed towards organ of pathology (eg. Mc Burney incision for appendicitis). Primary disease is then treated for example closure of a perforation, resection and anastomosis if the diseased segment is large, appendectomy in case of ruptured appendix.



A case of duodenal perforation



A case of intraperitonial abscess



A case of ileal perforation

A case of appendicitis

Peritoneal lavage: [7, 16]

A peritoneal wash is usually warranted in generalized peritonitis. Copious amounts of warm isotonic crystalloid solutions are used to remove gross particulate matter as well as blood and fibrin clots and reduces the bacterial load. Inclusion of antibiotics and antiseptics to the irrigating solutions is generally don't serve the purpose or even harmful as they may cause adhesions. Antibiotics given parenterally usually attain bactericidal levels in the peritoneal fluid thus adding them to lavage fluid will be unnecessary. After lavage the remaining fluid should be aspirated completely as it may later dilute the

opsonins and hamper the defense mechanisms and sometimes it get infected to form pelvic abscess or sub diaphragmatic abscess.

Peritoneal Drainage:

Use of drains in peritoneal cavity is topic of debate as there is a chance of introduction of more infection, the drains are sealed off early and may even predispose to abscess and fistula formation. If there is generalized peritonitis drains can be used, but in cases with localized peritonitis drains can be avoided. When used, closed drains should be used.

Post-operative care

Intensive care with ventilatory support may be needed especially for unstable and debilitated patients. Immediate objective is to achieve hemodynamic stability and adequate perfusion of major organs .Inotropics may be used. Antibiotics are given for 10-14 days depending on the severity of peritonitis. A favorable response is shown by maintained perfusion and adequate urine output, reduced fever and leukocytosis, resolution of ileus, and returning of sense of wellbeing of patient. Early removal of non-essential catheters is recommended. Early mobilization of patients helps in prevention of deep vein thrombosis and returning of sense of wellbeing of patient. Early

enteral feeding is advised which has the advantage of improving the sense of wellbeing as well as restore the gut flora.

Complications

Post-operative complications are frequent and can be divided into local and systemic complications. Deep wound infections, residual abscesses and intraperitonial sepsis, anastomotic breakdown, and fistula formation usually manifest by first week. Persistent fever, hypotension, generalized edema, abdominal distension, prolonged mental apathy may be the sole indicators of persistent intra-abdominal sepsis.

Uncontrolled sepsis leads to multi organ failure and ultimately death of the patient.

Prognosis

Overall mortality of generalized peritonitis is about 40%. Factors contributing to mortality will be studied in detail in our study through Mannheim peritonitis index scoring

REVIEW OF THE DIFFERENT SCORING SYSTEM

Different scoring systems have been developed over the years to try to accurately predict morbidity and mortality in patients requiring emergency surgical and medical care.

THE APACHE SYSTEM:

In 1981, Knaus and others proposed a scoring system to be used for classifying patients admitted to intensive care units. It consisted of two parts:

1. A physiology score representing the degree of severity of acute illness (The Acute Physiology Score)
2. A preadmission health evaluation indicating a patient's health status before the acute illness.

The Acute Physiological Scoring was developed using a panel of multidisciplinary physicians who selected laboratory and clinical measurements important inpredicting mortality. They restricted the selection to physiological variablesthat were available or obtainable on or shortly after admission to an ICU.Relative weights of importance were assigned so each variable was weighted onthe basis of its degree of abnormality and its relative importance compared with all its other measurements. Each physician in the group was free to suggestadditions or deletions of variables included on an initial list.

Ultimately, thepanel agreed on a list of 34 physiological measurements, and relative weights of importance were assigned on a scale from 0 to 4. The weights are neither symmetrical around the normal range nor uniform across different physiologicalmeasures.

In the original APACHE system, the greatest degree of abnormality for each physiological variable recorded within the initial 32 hours after ICU admission was used to create the score. Although 32 hours did allow for potential effect of therapy on physiology to be introduced, it provided time forall potential data to be available. The original APS for a patient was the totalpoints for all 34 variables.The second part of the original APACHE was the health questionnairethat assessed health status before admission. On the basis of answers toquestions regarding 1) number of recent visits to a physician, 2) work status,3)activities of daily living and 4) presence of carcinoma, a patient was given apre- ICU admission classification ranging from `A` for excellent health and `D`for severe failing health. The end result of APACHE was a separate APS andchronic disease classification for each patient. (E.g.: 14D, 16C etc.)

THE APACHE II SYSTEM: [17, 23]

The APACHE II system is a revised version of the original APACHE and was published in1985. The number of physiologic measurements was reduced from the original 34 to 12. Infrequently measured physiologic variables such as serum osmolality, lactic acid level, and the skin testing forenergywas deleted, as were potentially redundant variables. Each variable was deleted based upon clinical judgment and then evaluated using a multivariate comparison of the original APACHE system with each proposed revision, the total R2and the correct classification rate for hospital mortality was used standards. The smallest number of variables that reflected physiologic derangement for all vital organ systems as well as maintained statistical precision was 12.

Age and severe chronic health problems reflect diminished physiologic reserve and hence they have been directly incorporated into APACHE II. Chronologic age is a well-documented risk factor for death from acute illness that is independent of the severity of disease.During the validation, it was found that three of the four chronic health classifications (B, C, and D) were associated with higher death rates, when age and acute physiologic derangement were controlled. However, only the most severe chronic organ system insufficiency or immunocompromised state (Class D) markedly influenced outcome. It was also discovered that non-operative and emergency surgery admissions had a substantially higher risk for death from their prior organ

system insufficiency than elective surgical admissions. This was probably because patients with the most severe chronic conditions were not considered to be candidates for elective surgery. Therefore non- operative or emergency operative admissions with a severe chronic organ system dysfunction were given an additional five points, while similar elective surgical admissions were given only two points. The maximum possible APACHE II score is 719. The problem with APACHE system is that it uses many investigative modalities which may be out of reach for a common man.

SEPSIS SCORE: [18, 14]

Developed by Elebute and Stober in 1983, this system divides theclinical features of the septic state into four classes to which they ascribed a subjective degree of severity on an analogue scale. The attributes were

1) Local effects of tissue infection,

2) Degree of temperature elevation,

3) Secondary effects of sepsis and

4) Laboratory data.

The possible range of scores under this system is 0 to at least 45, depending on how the tables are interpreted. This system has been examined in detail by Dominions and associates. They reported on 135 patients with broad

variety of infectious problems, including peritonitis, pneumonia,wound infection, urinary tract infection, abscess, septicemia and mediastinitis. The sepsis scores ranged from 10 to greater than 30. In a group of patients with an overall mortality rate of 56%, they observed deaths of 13 of 64 patients (20%) with scores of 20 or below and 63 of 71(89%) with scores greater than20. if a score of 20 is arbitrarily chosen as a point above which death is predicted, the overall accuracy for this prediction will be 114 of 135 (84%).

PERITONITIS INDEX ALTONA:

Teichmann and associates, in a report concerning scheduled reoperation for diffuse peritonitis, referred to this index. In this study, they observed that mean peritonitis index for patients who died was 1.59, whereas that for patients who lived was 0.38. This index uses age, extent of infection, malignancy, cardiovascular risks, and leucopenia, to stratify patients

POSSUM: [17, 22]

Physiological and Operative Severity Score for enumeration of Mortality and morbidity (POSSUM) and its Portsmouth modification (P-POSSUM) were developed to provide risk-adjusted analysis in patients undergoing surgery. It consists of two parts:

Physiological assessment:

It provides exponential score on 12 variables. The physiological variables are: age, cardiac signs, respiratory signs, systolic blood pressure, pulse, coma score, serum urea, sodium, potassium, haemoglobin, white cell count, and ECG.

Operative severity:

- operative magnitude
- number of operations within 30 days
- blood loss- peritoneal contamination
- presence of malignancy
- timing of operation

THE SIMPLIFIED ACUTE PHYSIOLOGICAL SCORE (SAPS):[12, 16, 21]

This system was developed by Le Gall et al in 1984 as an independent attempt to simplify APACHE. It was a European north American study undertaken from September 1991 through February 1992. Patients were enrolled from September 1991 through December 1991. Totally 13152 patients were enrolled from 10 countries from different hospitals. Patients were followed up for 2 months and any patient remaining in hospital after February 28 1992 was dropped from the study. All consecutive admissions, 18 year or older, to adult ICU in the participating hospitals were eligible for enrollment,

but burns, coronary care patients, and cardiac surgery patients were excluded. After data collection the validity of data was inspected by checking randomly by a second person. Data was collected for the first 24 hours of admission. To develop the scoring 65% of patients were selected as developmental data set and 35% as validation data set. For each variable LOWESS smoothening function was used to suggest ranges for each variable. For assigning points for each variable, dummy variables were created and multiple logistic regression analysis was used and resultant coefficients of this analysis were used to assign the points to the ranges. The points were multiplied by 10 and rounded off to the nearest integer. Hosmer-Lemeshow goodness of fit test were performed on both developmental and validation sets to assess the performance of the system. An expected outcome within each decile of population was compared with actual outcome to assess the goodness of fit. Out of 37 initial variables selected using multiple regression technique 13 variables which individually affected the prognosis of the patient were selected.

It found that distribution of SAPS was highly skewed. Thus an integration was used. Thus the equation had to accommodate SAPS II and In[SAPSII +1]. Using these logit was calculated as

Logit = -7.7631+{0.0737x(SAPSII)}+{0.9971xln[(SAPSII)+1]}

this logit was converted to hospital mortality was calculated using following equation

$$\text{Predicted Mortality} = e^{(\text{Logit})} / (1+e^{(\text{Logit})})$$

The Reviewer Observation Characteristics (ROC) curve was plotted for SAPS II was calculated and the area under ROC was found to be 0.88 (95% confidence interval).

JABALPUR SCORING FOR PEPTIC ULCER PERFORATION

It's a simplified system of scoring in patients with duodenal perforative peritonitis.It doesn't need sophisticated laboratory values. It can be used in peripheral hospital were no ICU setup .The scoring considers age,sex,perforation to operationinterval, comorbid disease, mean systolic pressure, heart rate, respiratory rate, Haemoglobin level, serum creatinine, chronic ulcer history. Score ranges from 0-21.

The multiple regression equation for complications and death were as follows:Predicted mortality score = 0.12+ (0.16xage) + (0.10 x perforation operation interval) + (0.12 x comorbid disease) + 0.06 x shock + (0.04 x heart rate – 0.27 x female sex)

Predicted complication rate = -.06 + (0.28 x preoperative shock)+ (0.27 x comorbid illness) + (0.23 x raised creatinine) +(0.21 x anemia) + (0-16 x respiratory rate score) + (0.19 x perforation – operation interval) +(0.14 x chronic ulcer history score)

Score between 15 to 21 has got increased morbidity and mortality.

MANNHEIM PERITONITIS INDEX [6, 7, 8,9,10]

The Mannheim Peritonitis Index was developed by Wacha and Linder in 1983. Itwas developed based on the retrospective analysisof data from 1253 patients with peritonitis, inwhich 20 possible risk factors were considered. Ofthese only 8 proved to be of prognostic relevanceand were entered into the Mannheim PeritonitisIndex, classified according to their predictive power.Patients with a score exceeding 26 were definedas having a high mortality rate. The MannheimPeritonitis Index is a specific score, which has agood accuracy and provides an easy way to handlewith clinical parameters, allowing the prediction ofthe individual prognosis of patients with peritonitis. The Mannheim Peritonitis Index is one of thesimplest scoring systems in use that allowsthe surgeon to easily determine outcome risk. Theircollection of retrospective data is possible andvalid, because Mannheim Peritonitis Index onlyrequires information routinely found in surgical registers.

MANNHEIM PERITONITIS INDEX [7, 8, 9]

Table 2

Risk factors	**Weightage, if any**
Age >50yrs	5
Female gender	5
Organ failure	7
Malignancy	4
Preoperative duration > 24 hrs.	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	
Clear	0
Cloudy	6
Fecal	12

Definition of organ failure

Creatinine> 2.0mg/dl,

Urea> 60mg/dl, oliguria<20ml/h;

Lung pO2<50mmHg; pCO2> 50mm Hg

Shock: hypodynamic or hyperdynamic;

Intestinal obstruction: paralysis >24hrs or complete mechanical ileus

MPI calculated at admission or during management by careful history taking and physical examination basic blood investigations which includes complete hemogram , Blood urea ,Serum creatinine ,ABG analysis , continous vital monitoring for sick patients. Patients followed up until discharge or death. Patients were grouped into three categories based on disease severity those with MPI less than 21, between 21 and 29, and ,more than 29. Patients with MPI > 29 have poor prognosis. The value of MPI score is it helps in assessing patients by aggregating several variables regarding severity of peritonitis preoperatively, intraoperatively and predict outcome of disease

STUDY DESIGN

This is an observational study

Number of patients: 100

Inclusion Criteria:

Patients with secondary peritonitis managed in surgical ward and ICU in Coimbatore Medical College Hospital during the period Sept 2012 to Sept 2013

Exclusion Criteria:

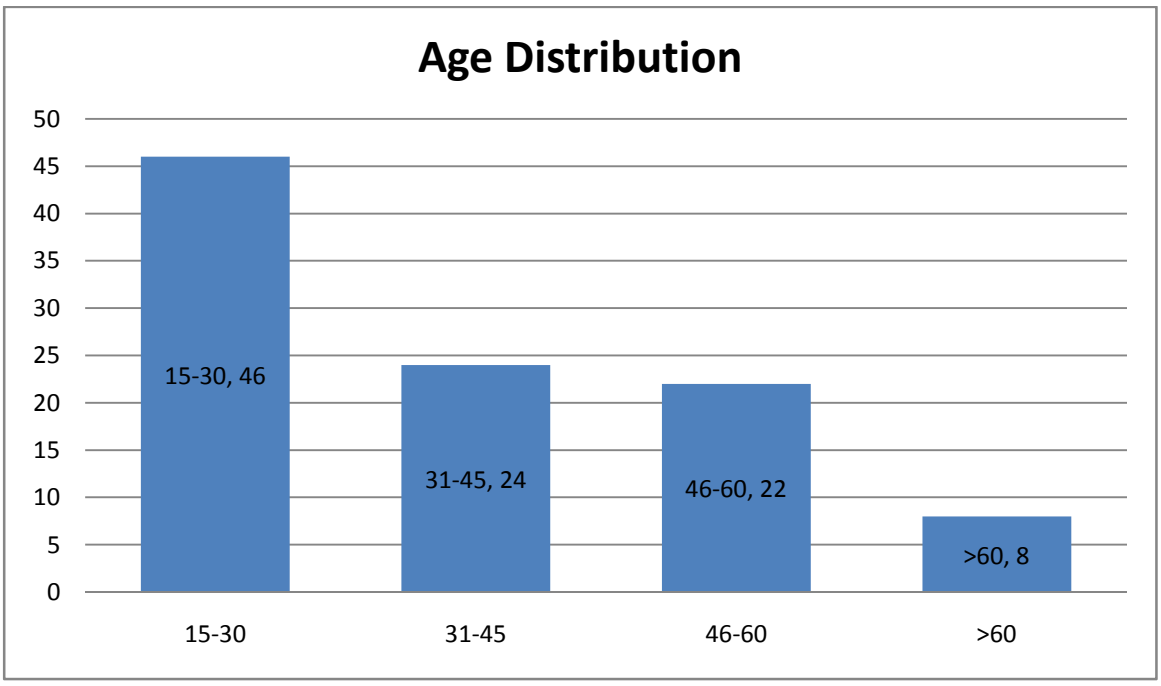
- Patients with primary peritonitis
- Spontaneous bacterial peritonitis
- Pancreatitis
- Intra-abdominal sepsis due to peritoneal dialysis

Methodology:

Resuscitation measures, antibiotic therapy, vasoactive drugs, nasogastric intubation and analgesics administered as required. MPI were calculated at admission or during management. All patients will undergo laparotomy and managed according to thecause. After surgery interventions like antibiotic therapy, vasoactive drugs, resuscitation and ICUcare given as necessary. Patients followed up until discharge or death. Patients are grouped into three categories based on disease severity those with MPI<21, between 21 and 29,> 29. Mortality rates calculated belonging to each group.

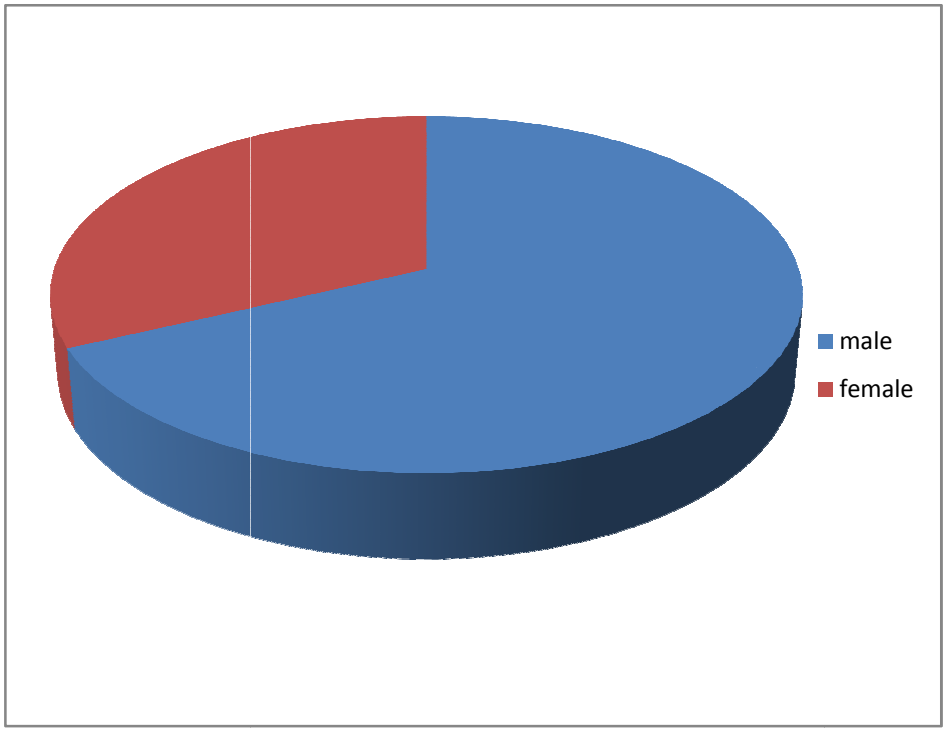
RESULTS

The Mannheim Peritonitis Index (MPI) was evaluated in 100 consecutive patients with both local and general peritonitis who admitted in surgical wards and SICU. MPI was calculated before and during the surgical intervention .Age distribution among these patients are



SEX DISTRIBUTION

Of 100 patients with peritonitis 68 were males and 32 were females



Mortality in the study group was found to be 25/100. 23 deaths were from male population. Females contributed 2 deaths.

Table No 3

MORTALITY

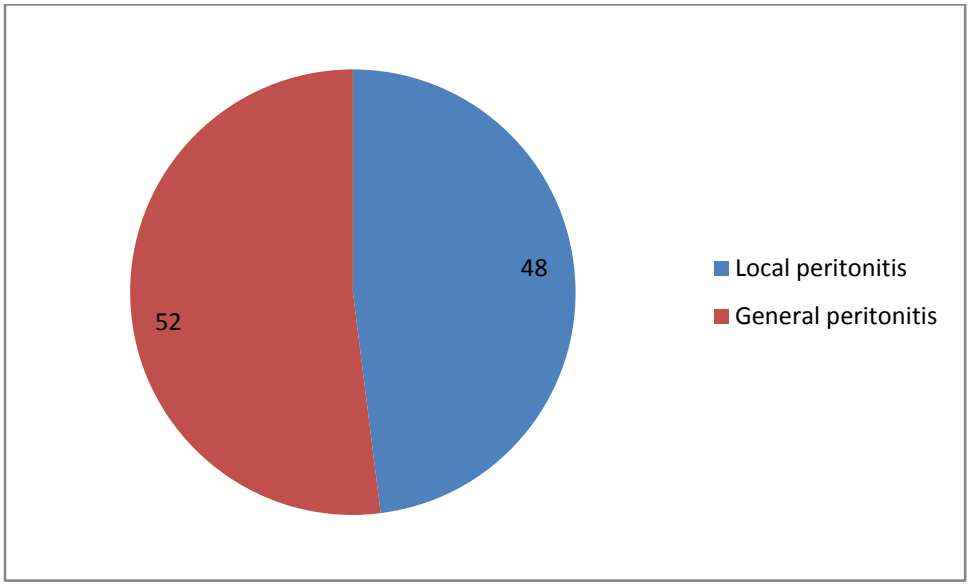
	Males	Females
Death	23	2

DISTRIBUTION OF PERITONITIS

Among these group of patients distribution of peritonitis is as given in table

Table 4

Local peritonitis	48
General peritonitis	52

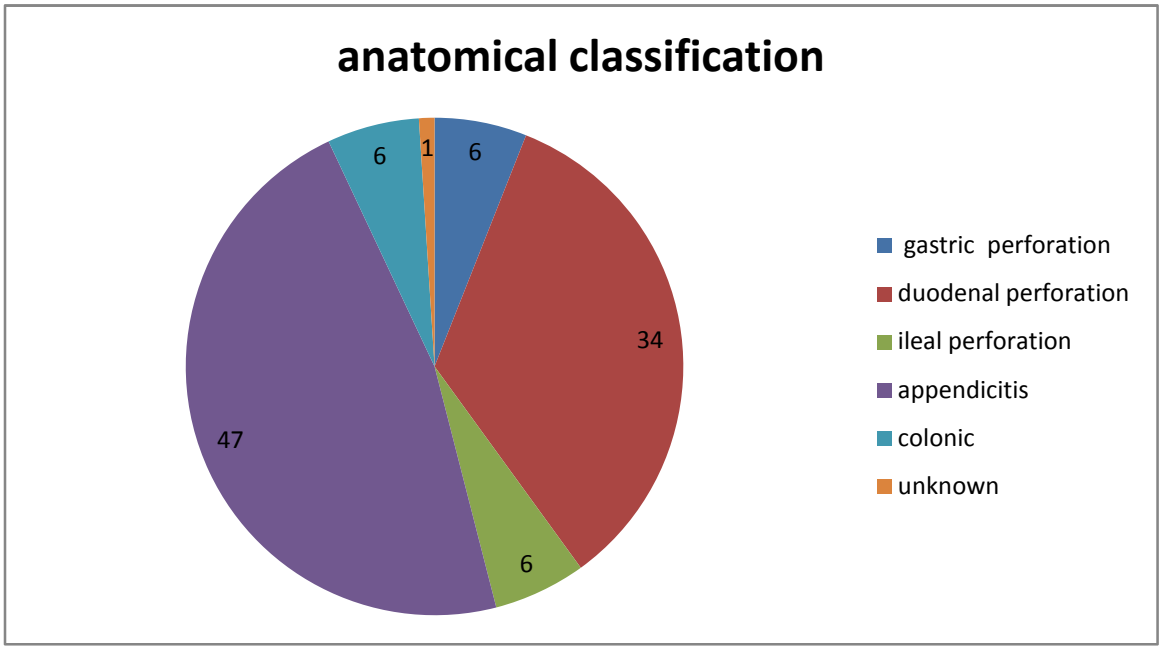


ANATOMICAL CAUSE OF PERITONITIS

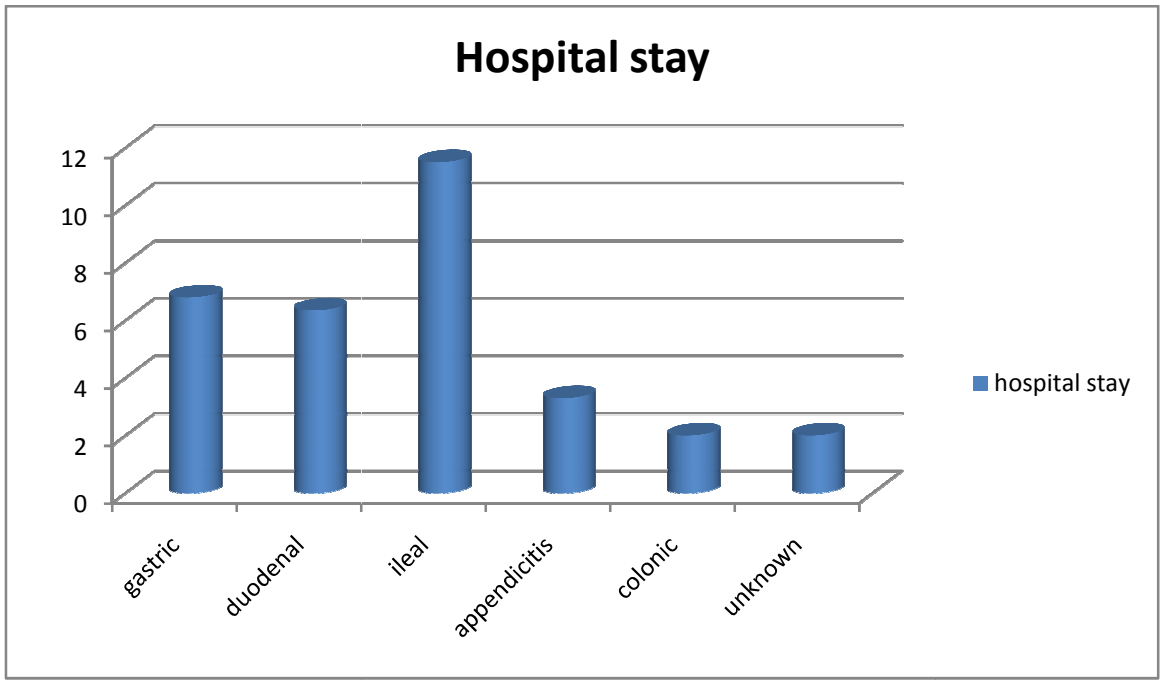
We classified patients according to the origin of peritonitis. The bulk of the local peritonitis was due to appendicitis (47%).Maximum cause of general peritonitis was due to duodenal perforation(34%) followed by colon(6%), ileum(6%), gastric(6%), and unknown (1%). The Cause was not made out for 1 patient in whom flank drain was put.

Table No 5

	Gastric	**Duodenal**	**Ileal**	**appendicitis**	**Colonic**	**Unknown**
Number	6	34	6	47	6	1
Death	4	12	2	0	6	1
Hospital stay	6.83days	6.35days	11.5days	3.3days	2.6days	2days



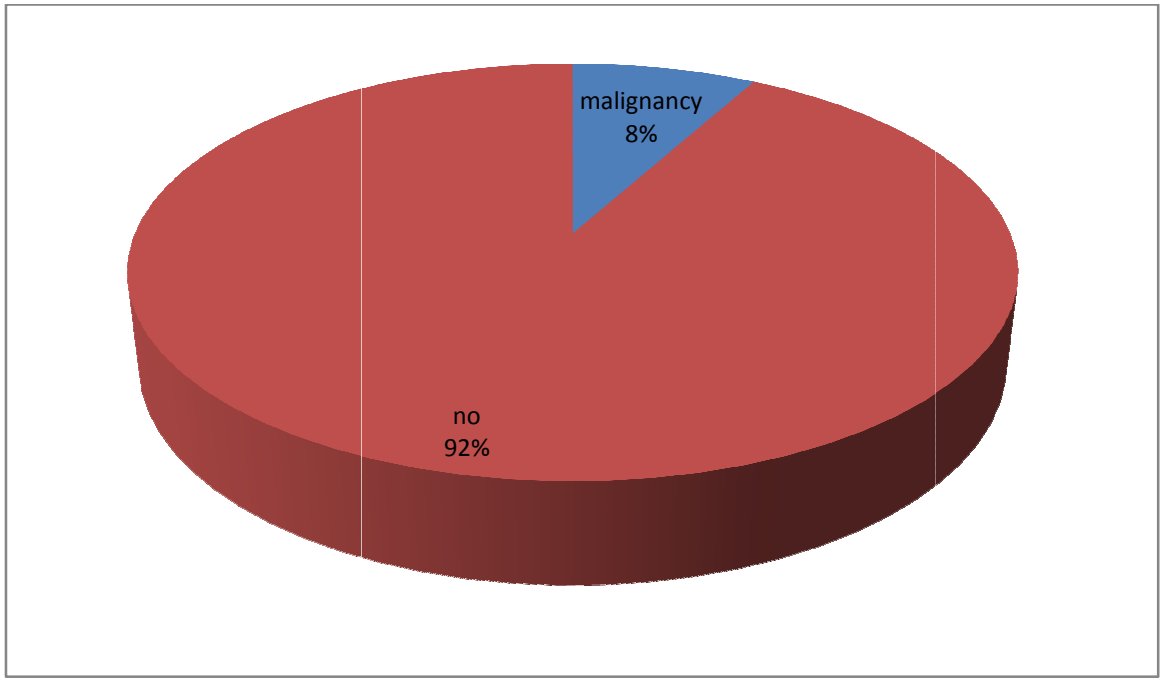
The patients having appendicitis have a lesser hospital stay and early recovery. Whereas highest hospital stay was found in the patients with ileal perforation followed by duodenal perforation and gastric perforation.



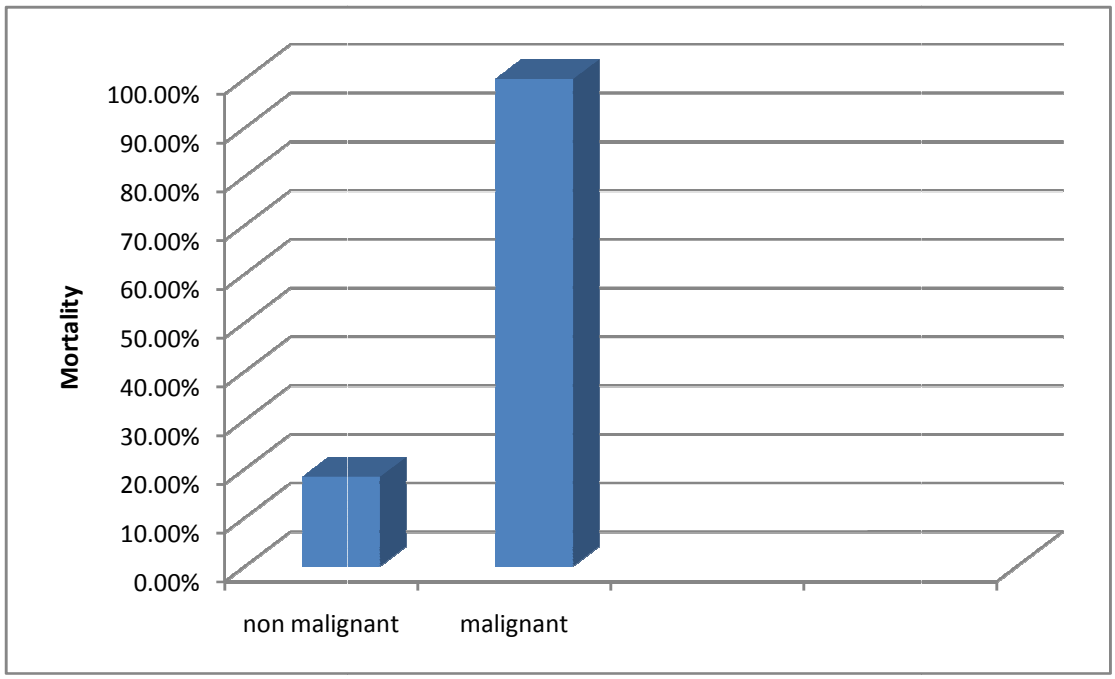
Colonic perforation has a lower hospital stay as all patients died in immediate post operative period.

PRESENCE OF MALIGNANCY

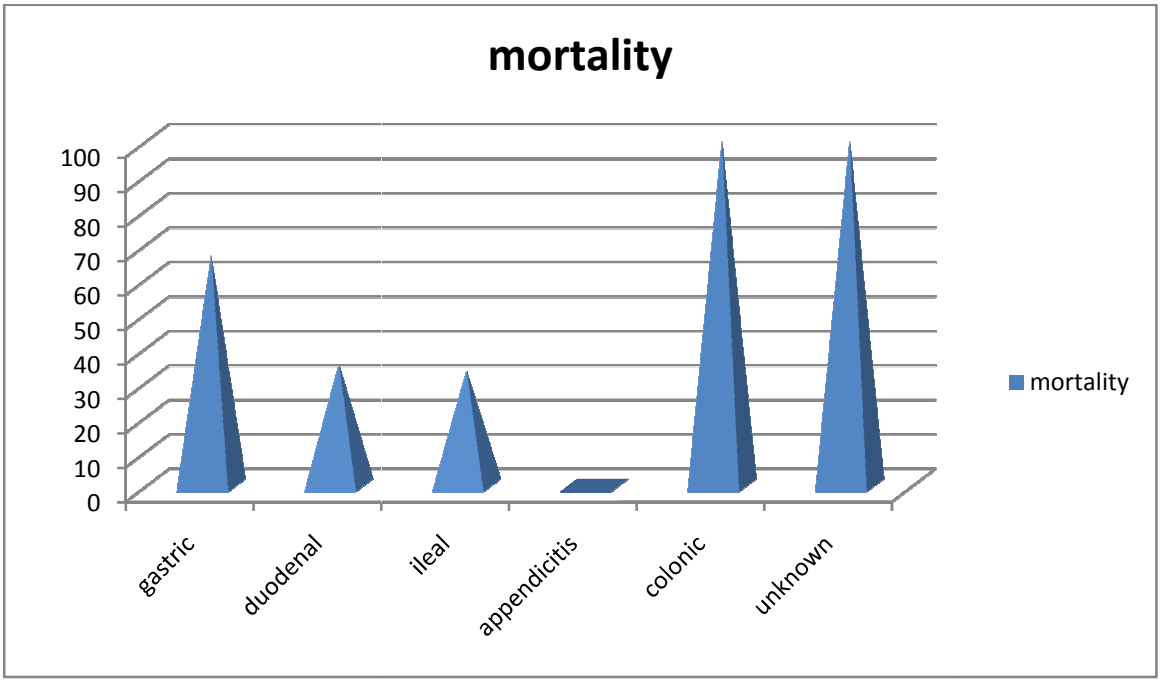
Among these8% patients was having malignancy. Perforation is eitherdue to closed loop obstruction and caecal perforation caused by malignancy or through malignancy.



All patients with peritonitis secondary to malignancy died due to late presentation and general fecal peritonitis



MORTALITY



The number of death in each type of perforation is shown in table and the mortality rate in each category is shown in the above chart. The highest mortality rate was found in the group of colonic perforation and unknown site of perforation followed by gastric perforation.

Though the appendicitis and duodenal perforations formed the bulk of the diagnosis mortality was relatively low. May be due to containment of peritonitis in case of appendicitis and comparatively earlier presentation in duodenal perforation

MORBIDITY ANALYSIS

The various complications were recorded. The most common complications were found to be wound infection and respiratory complications followed by the urinary tract infections and enterocutaneous fistulas. Intra-abdominal abscesses were found to be less in number.

Table No 6

	WI	**WD**	**RC**	**UTI**	**IAA**	**ECF**
Number	25	5	22	10	5	5

WI- wound infection WD- wound dehiscence

RC- respiratory complications UTI- urinary tract infections

IAA- intra abdominal abscess ECF- enterocutaneous fistula

Complications in individual anatomic sites

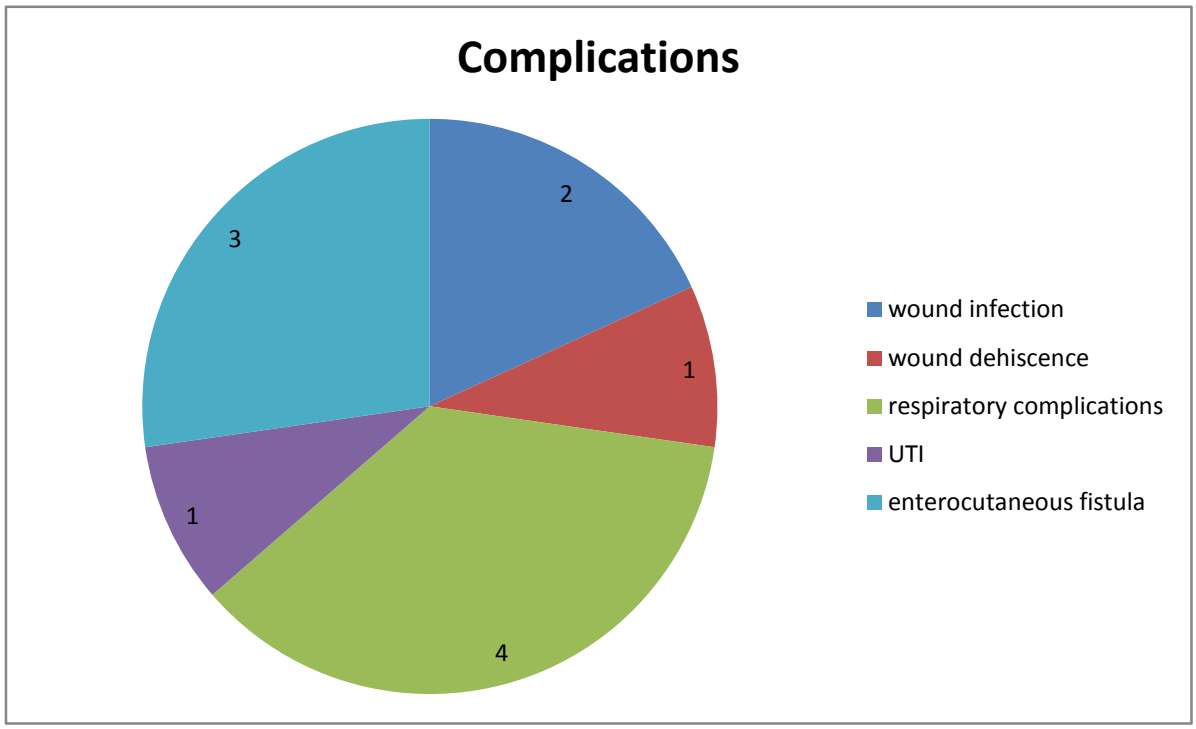
The complications were found to be highest in the colonic perforations. Followed by gastric perforation

Wound infection was found to be highest in duodenal perforations whereas respiratory complications were highest in the duodenal perforations. 3 cases who had intra-abdominal abscess were both having duodenal perforations. Enterocutaneous fistula was found to be higher in gastric perforation.

Table No7

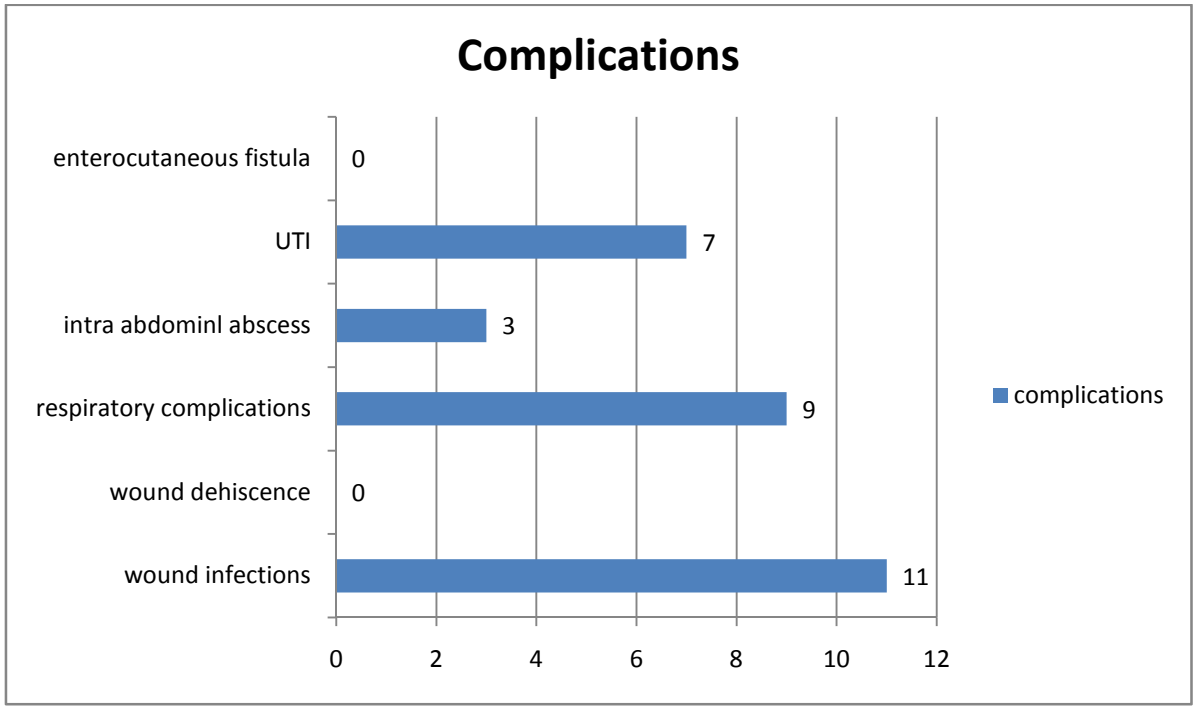
	WI	WD	RC	IAA	UTI	ECF
Gastric	2	1	4	0	1	3
Duodenal	11	0	9	3	7	0
ileal	5	1	4	1	2	1
appendicitis	5	3	0	0		1
Colonic	2	0	4	1	0	0
flank drain	0	0	1	0	0	0

GASTRIC PERFORATION



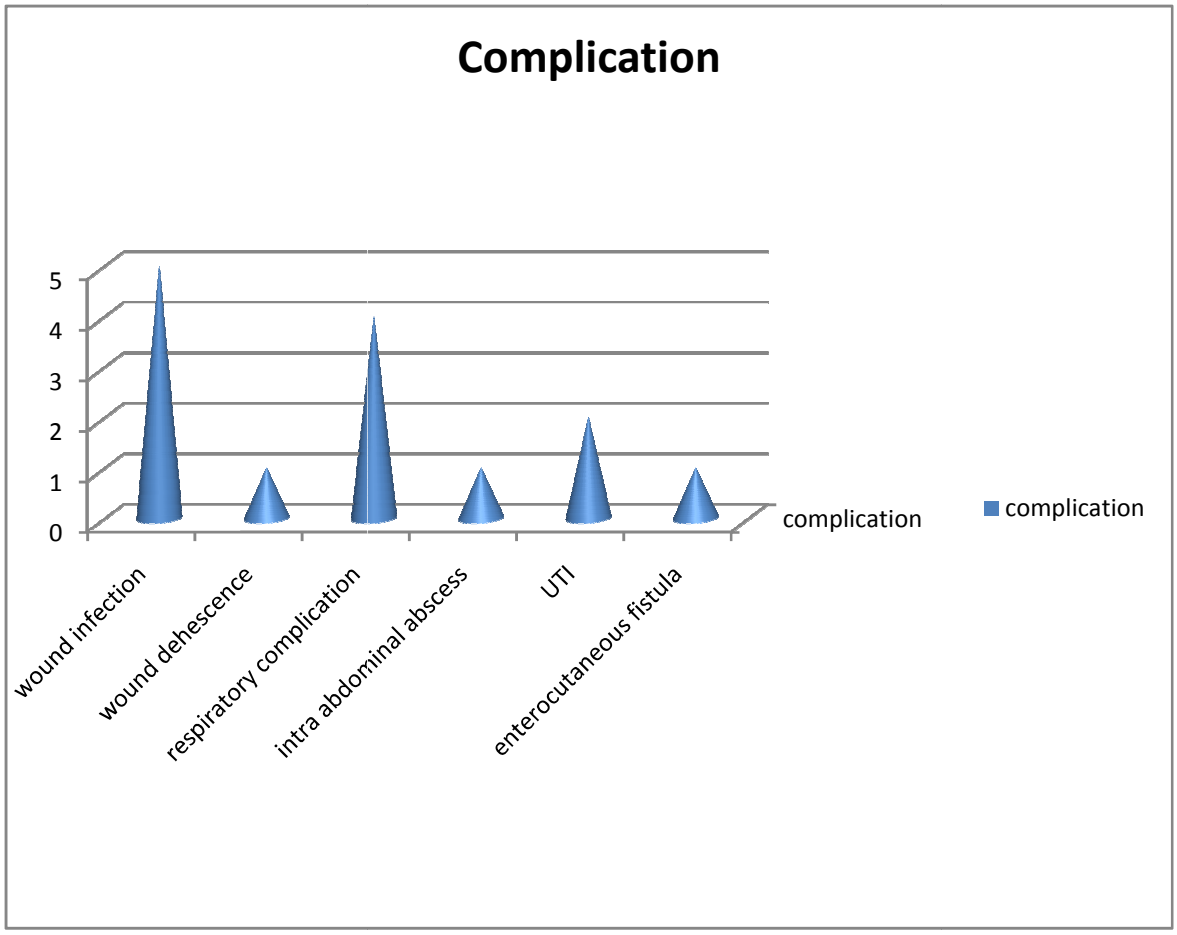
Most common complication in gastric perforation was respiratory complication followed by enterocutaneous fistula and wound infection.

DUODENAL PERFORATION



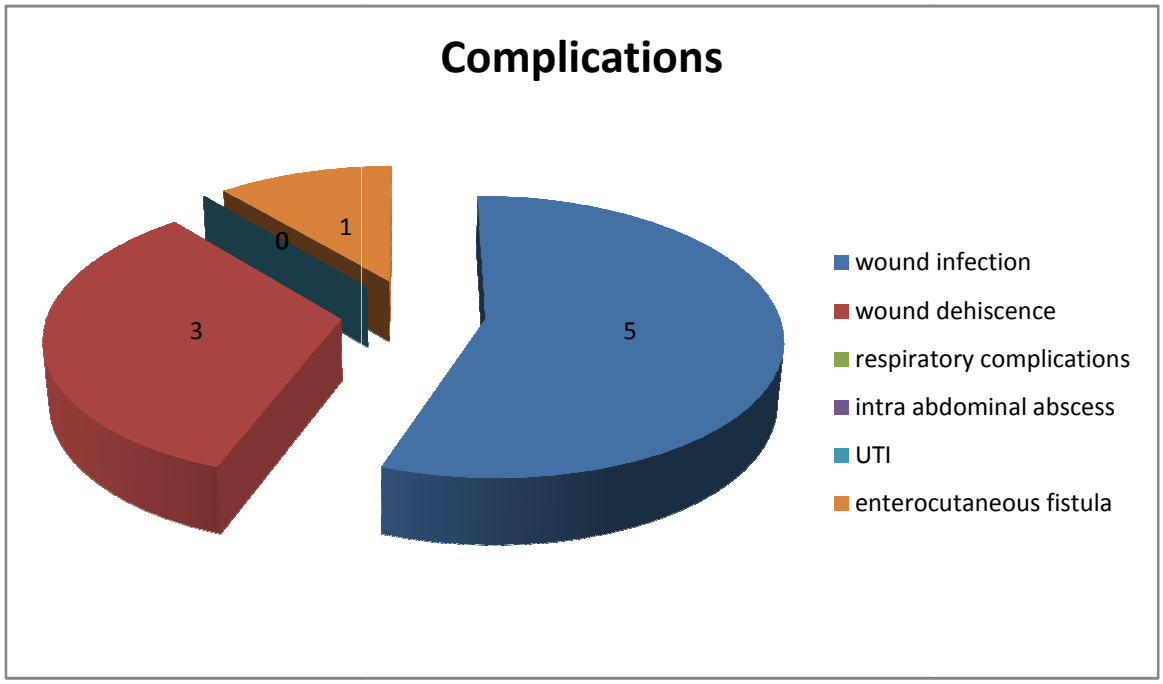
Wound infection and Respiratory complications were the most common complication found in duodenal perforation followed by urinary tract infection and intraabdominal abscess there were no cases ofenterocutaneous fistulas and wound dehiscence.

ILEAL PERFORATION



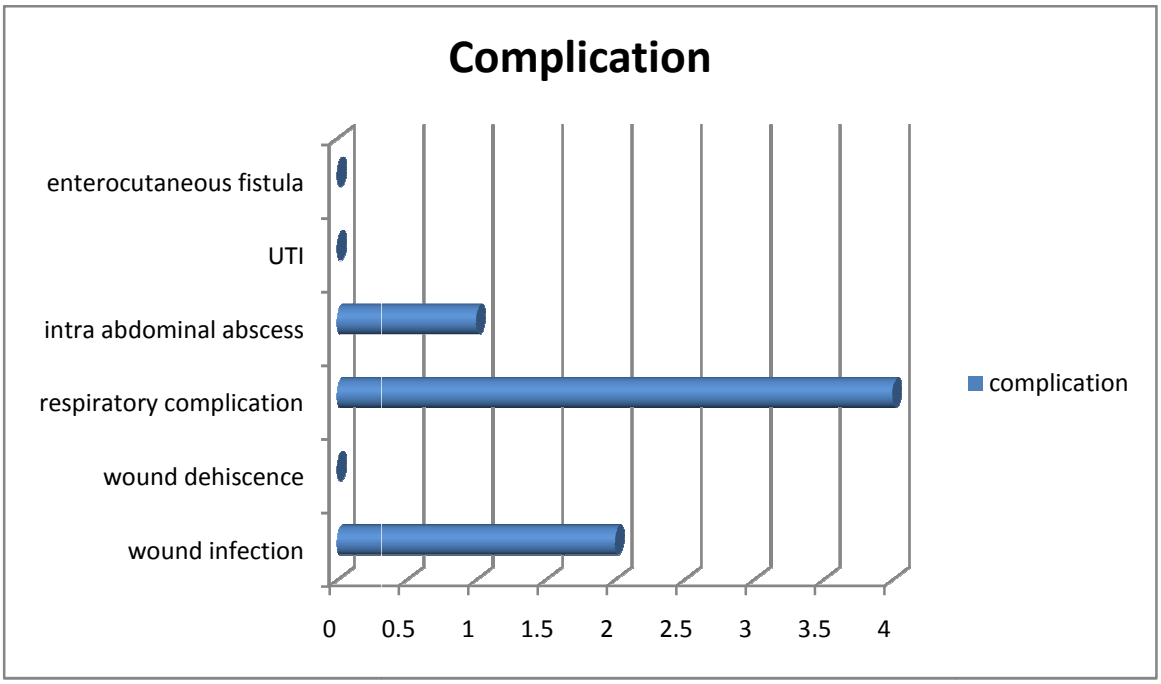
Wound infection is the most common complication in ileal perforations followed by respiratory complications and urinary tract infection. One patient found to have Intra-abdominal abscesses. One patient developed wound dehiscence and one patient developed enterocutaneous fistula.

APPENDICITIS



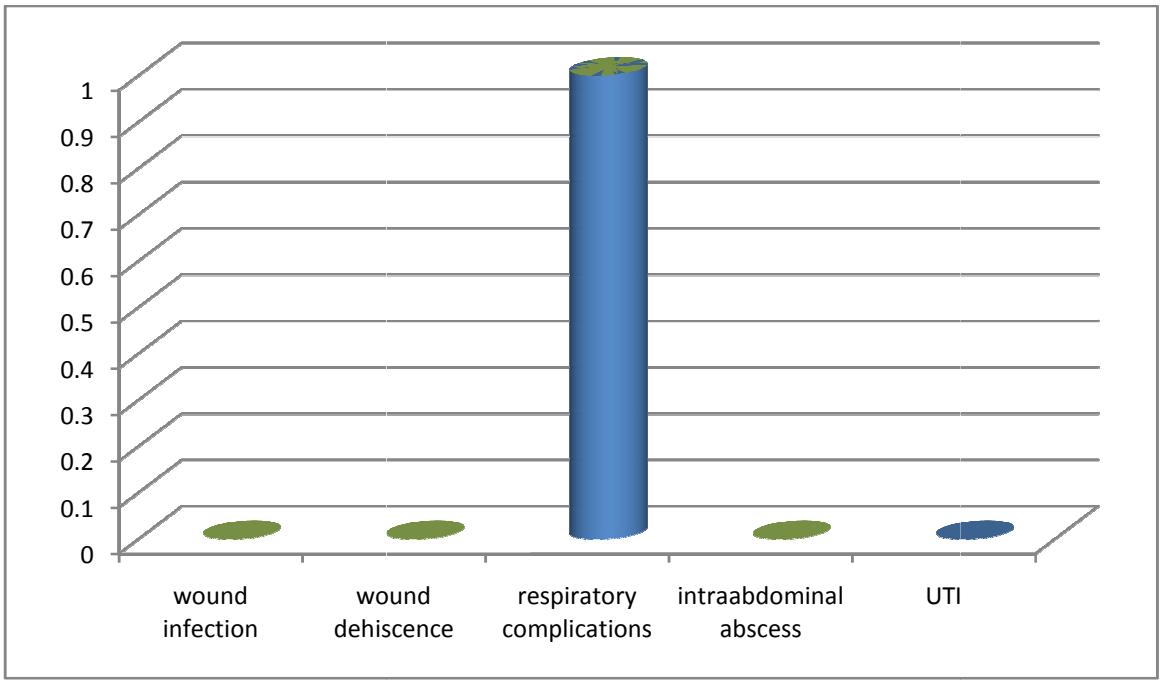
Wound infection were the most common complication in appendicitis followed by wound dehiscence, and enterocutaneous fistula. Enterocutaneous fistula was managed conservatively.

COLONIC PERFORATION



Complications were high in patients with colonic perforations. Two patients washaving wound infectionand four patientswas having respiratory complication,one patientdeveloped intra-abdominal abscess. Less number of complications are due to patients with colonic perforation succumbs to death immediate postoperative period.

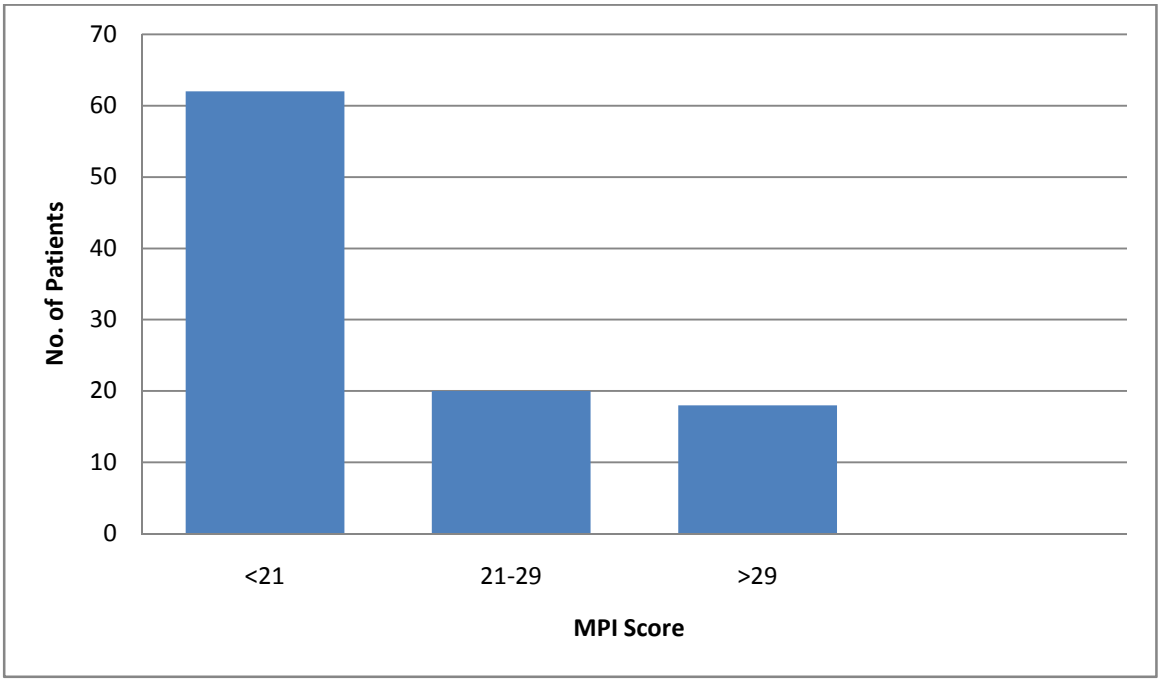
PERITONITIS DUE TO UNKNOWN ORIGIN/ FLANK DRAIN



Flank drain was done for one patient only. That patient expired immediate post-operative period patient was having respiratory complication as a part of multi organ failure

CALCULATED MPI SCORES

Mannheim peritonitis index was calculated in patients with peritonitis preoperatively and during the surgical procedure. Calculated MPI scores given as chart below



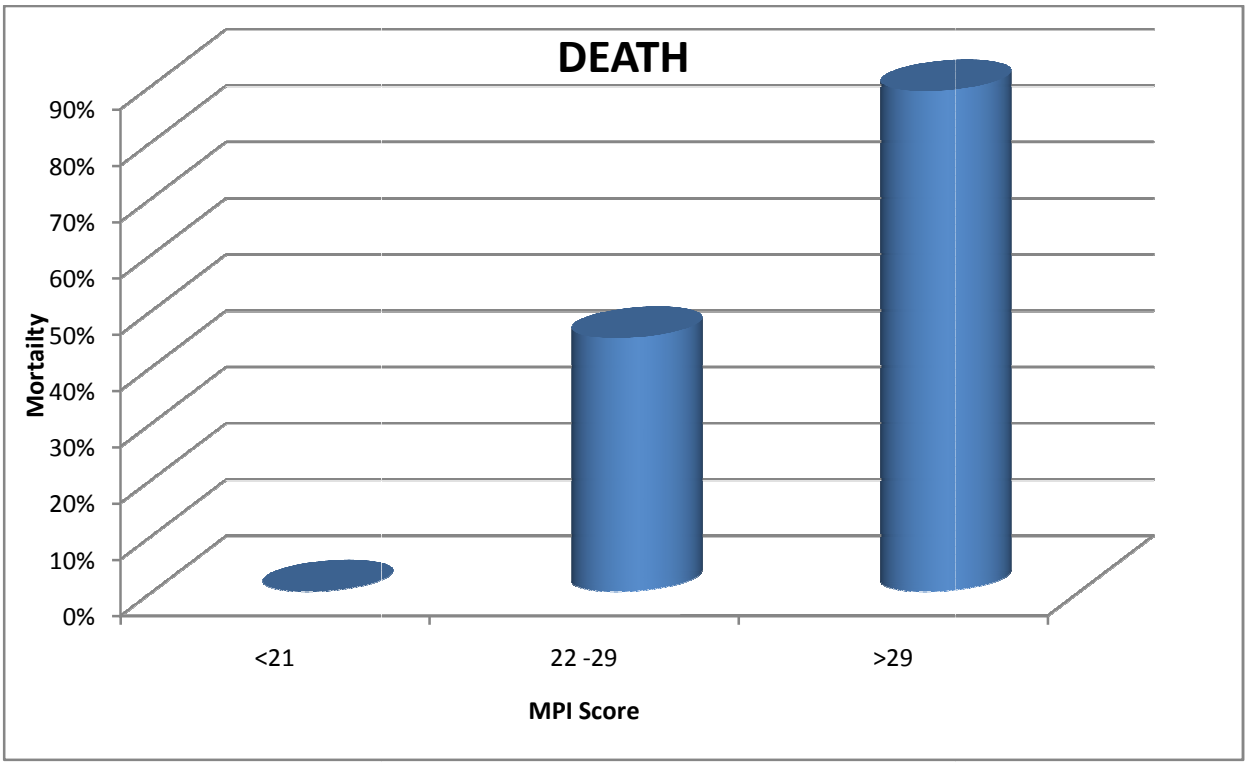
The patients with peritonitis is categorized in three groups .first group score less than 21 was managed by appropriate surgery (for example appendectomy for appendicitis , laparotomy for duodenal perforation) Usual care was given in postoperative ward for this patient

For patients with score between 21 and 29 and more than 29 was taken for ta surgery as early after stabilizing hemodynamic ally. Given intensive care by continuous monitoring of vitals postoperatively. Daily monitoring of renal function tests was done .patients was given higher generation antibiotics such as

Piperacillin –Tazobactum wasgiven. Ventilator support, inotropic support and intensive care as needed.

Due to active intervention in group of MPI score less than 21 mortality rate is 0Patients with MPI score between 21 and 29 was grouped under moderate risk.They were managed actively early .so mortality rates is 45%. Patients with MPI score more than 29 was grouped under higher risk group They were also managed early and intensive care was given as needed, but mortality rates was approximately 89 %

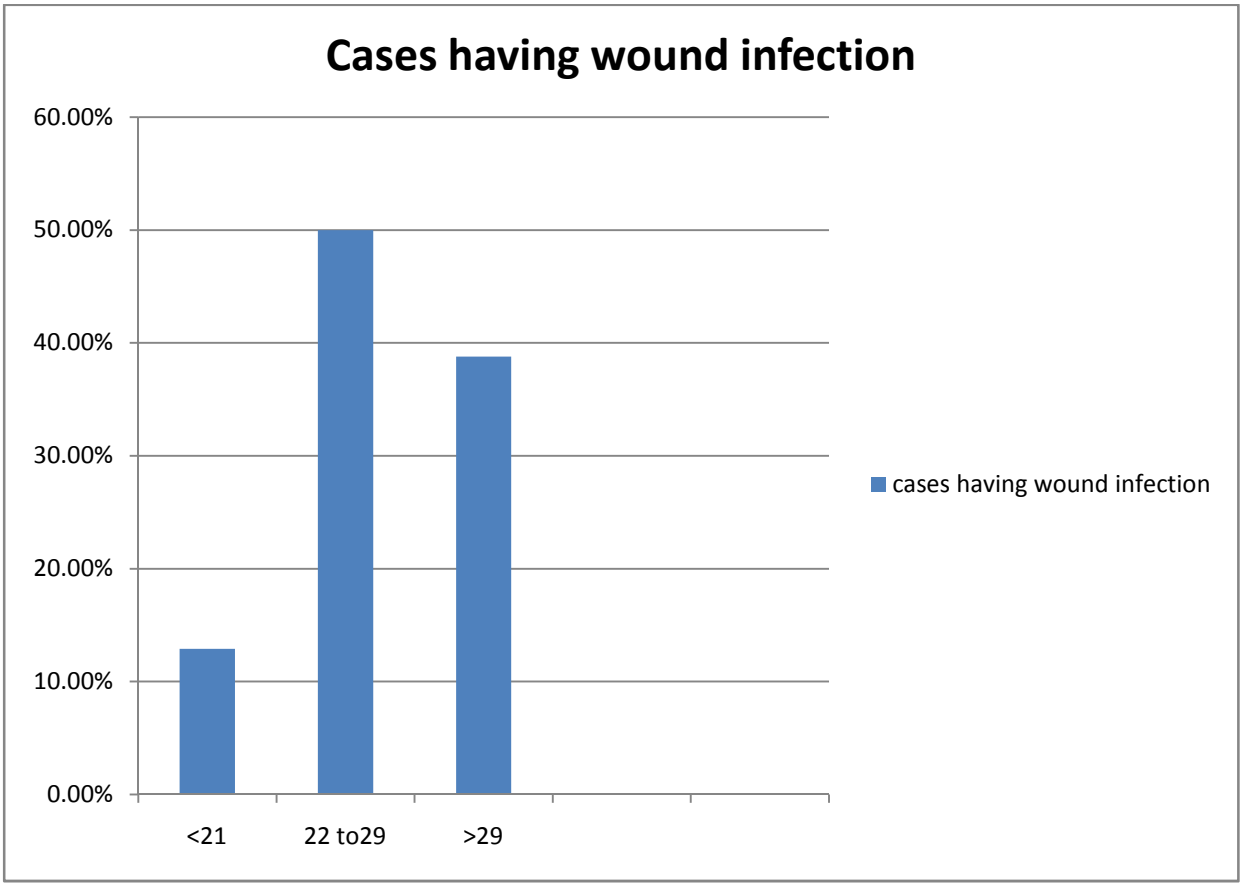
MORTALITY



Patients with MPI score <21 has 0%, score between 22-29 has mortality 45% and score more than 29 has got maximum mortality of 90%

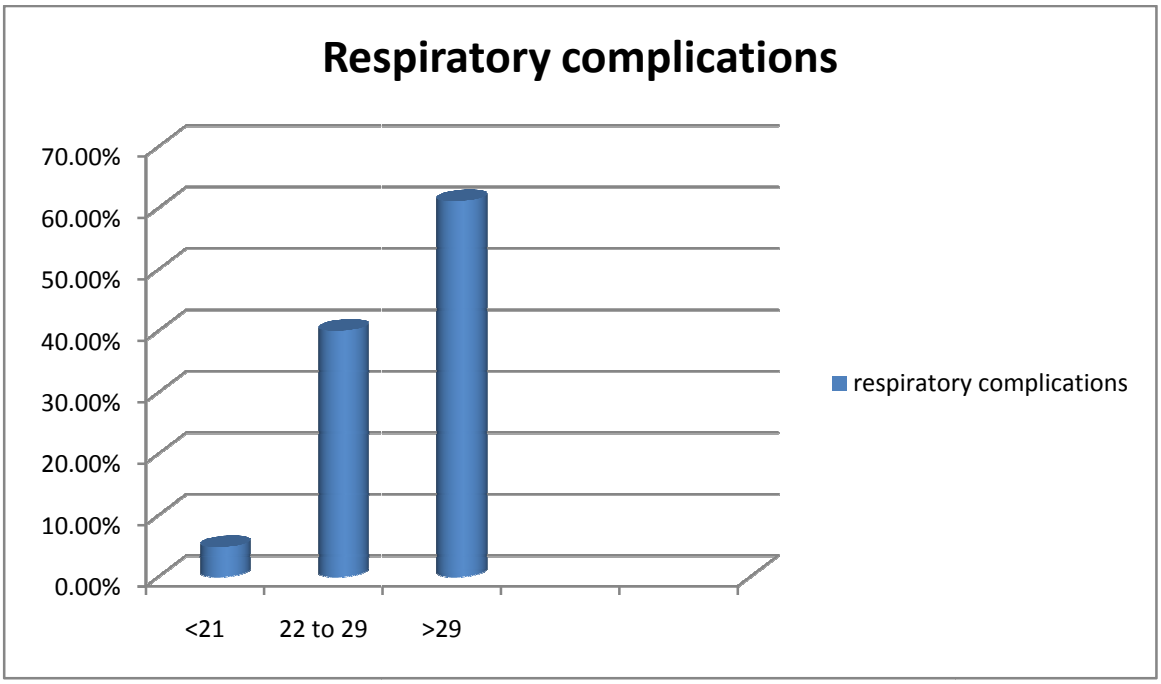
ANALYSIS OF COMPLICATIONS WITH MPI SCORING

WOUND INFECTION



When we evaluate the incidence of wound infections according to MPI score we find that when MPI score is less than 21 the wound infection rate is very low whereas with higher MPI scoring there is an increased wound infection rate. The wound infection rate in cases with MPI above 29 is lower than those with 22 to 29 as many cases died in early post-operative period before developing wound infection.

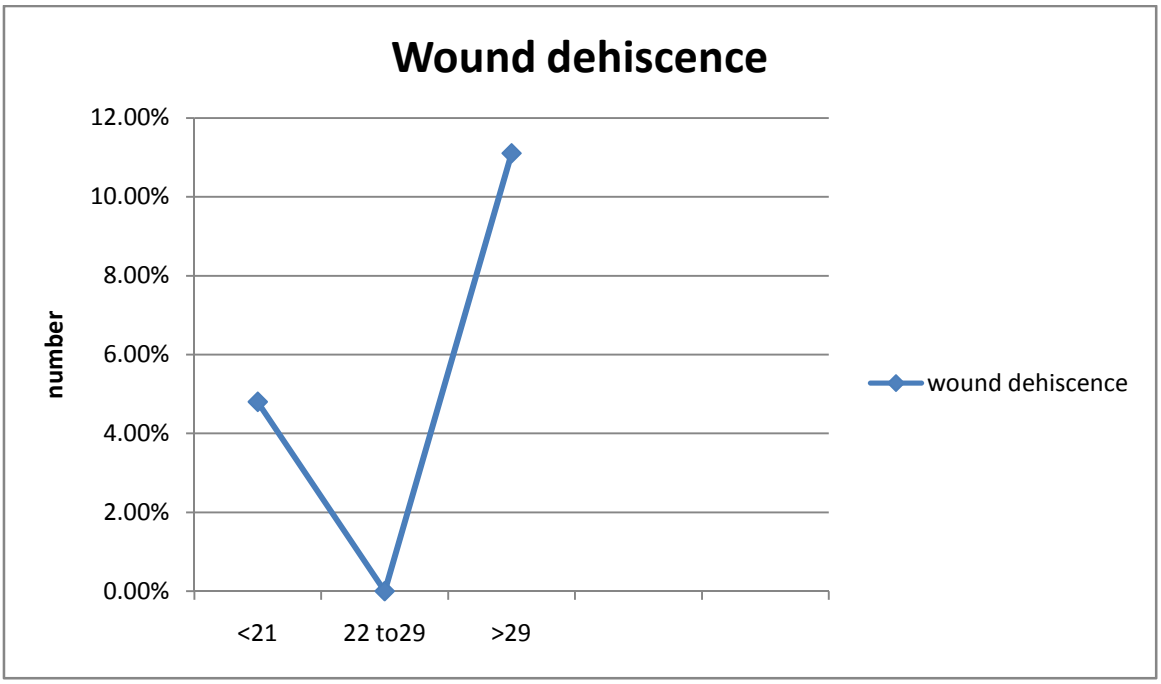
RESPIRATORY COMPLICATIONS



Respiratory complications had a linear relation with MPI scoring. Respiratory complications were actually the cause for most of the deaths.

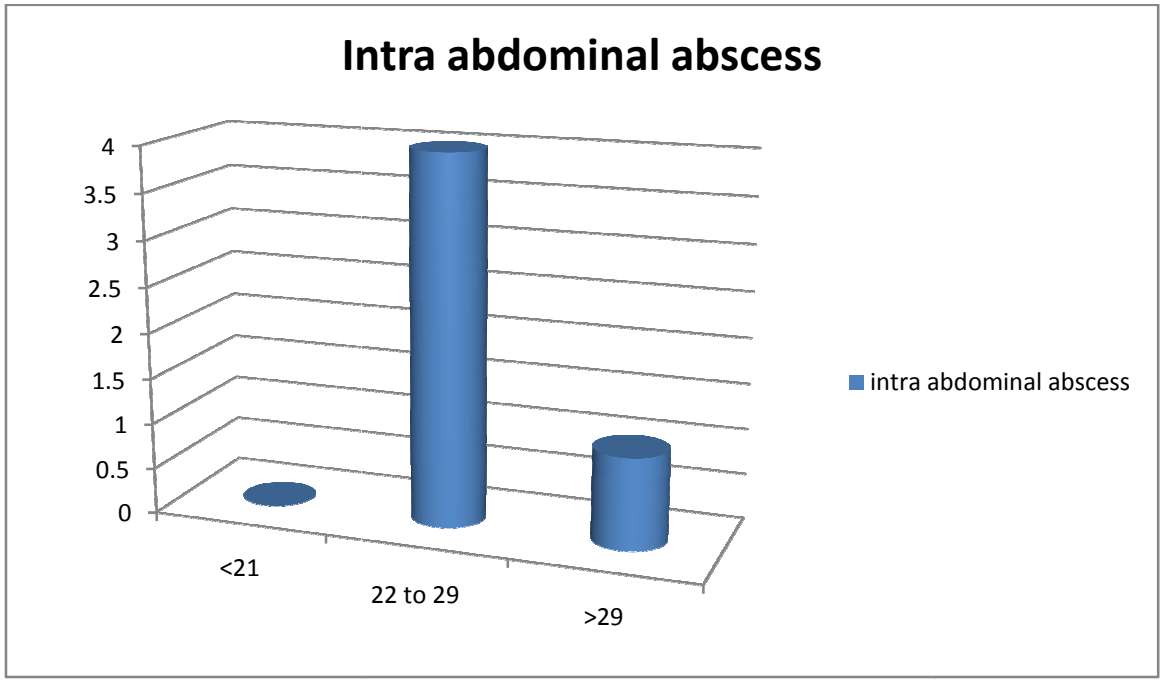
Thus we see 61.1% of patients with MPI scoring between 22 and 29 and more than 29 developing respiratory complications.

WOUND DEHISCENCE



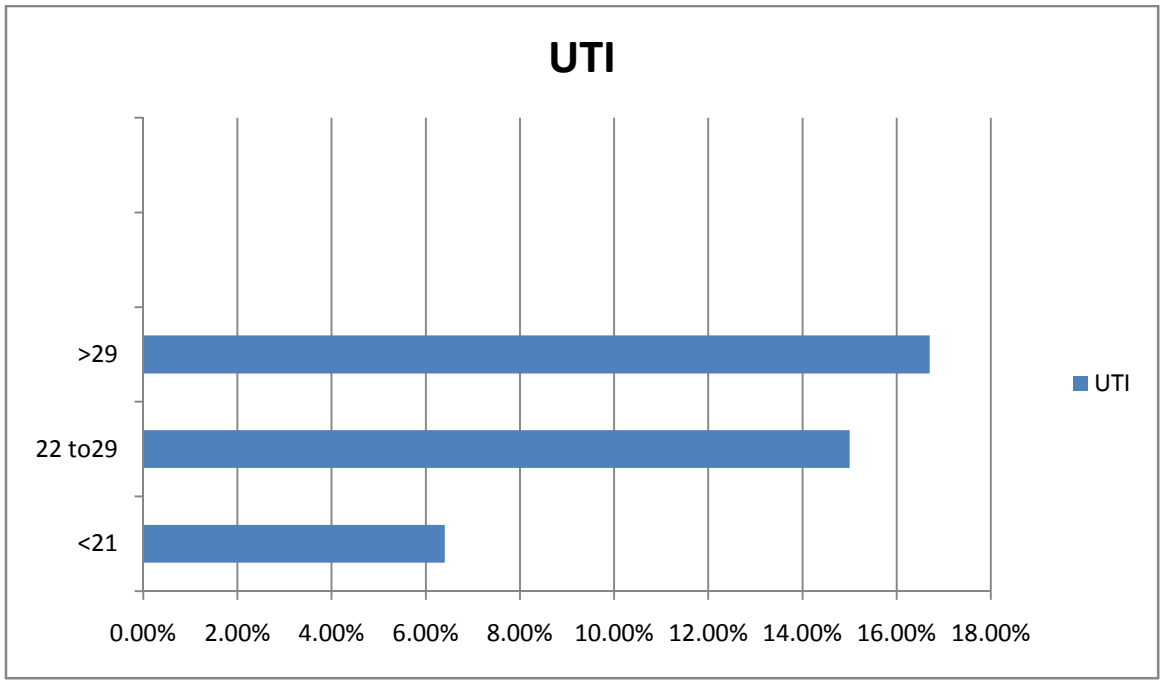
This chart shows the increasing cumulative cases having wound dehiscence as the MPI scoring increases. A MPI score below 21 has less wound dehiscence whereas when it is more than 29, 11.1% cases have wound dehiscence. No wound dehiscence in patients with score between 22 and 29

INTRA-ABDOMINAL ABSCESS



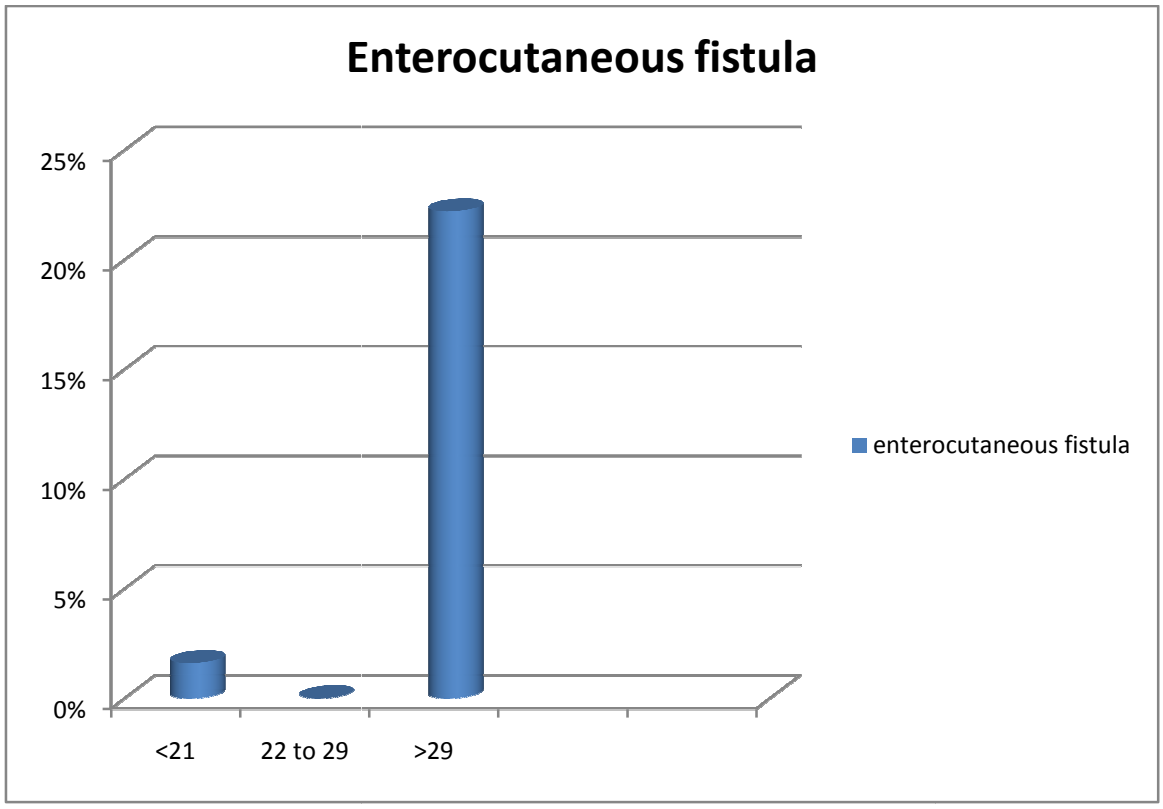
Of the 5 cases having intra-abdominalabscess, four of them came under the group having MPI score as 22 to 29 and one in patient with MPI score >29 . It was totally absent in cases having a score below 21

URINARY TRACT INFECTIONS



Urinary Tract Infections were almost uniformly distributed throughout the range of MPI scoring. This was probably due to almost universal use of bladder catheters in patients and indwelling catheter being a single most common risk factor for developing UTI.

ENTEROCUTANEOUS FISTULA



Enterocutaneous fistulas had the similar distribution to the other complications, being highest in the more than 29 range and being very less below a MPI score of 21.this complication was absent in group with score between 22 and 29

DISCUSSION

Peritonitis is the most common surgical emergency attended in a hospital. It can localized or generalized type of peritonitis. Even though intensive care and sophisticated investigative tools helps in managing this patient, it is the major cause of morbidity and mortality

Our study included 100 patients, in which showed amale predominance. The ratio is approximately 2:1

Most of the patients belong to age group between 15 to 30.

Most common etiology is acute appendicitis followed by duodenal perforation. The perforation of proximal gastro intestinal tract is most common as compared to western statistics which site at lower gastrointestinal tract. Major cause of post-operative morbidity was wound infection and respiratory complications. This is corresponding with the results of various other studies on peritonitis.

Duration of hospital stay doesn't correlate with severity of disease because a patient with MPI score more than 29 succumbs to death in immediate postoperative period.

The complications have been most common in the group of patients having a MPI score between 22 and 29, whereas those who have a score above 29 have higher mortality.

The result of this study is similar when compared to different studies conducted for peritonitis.

The scores below 21 has got good prognosis and mortality is 0% in this group. Patients with scores between 22 and 29 the mortality was 45%

Patients with scores morethan 29 the mortality was 90%. These correlates with study conducted byAli Yaghoobinotashetalpublished in Indian society of Gastroenterology with results of 0%, 60%, 90% mortality in patients with score <21 , 22-29 ,> 29 respectively

Study published by C G Nwigweetalin Ebonyi Medical Journal shows MPI score more than 30 has got increased mortality. MPI score of 25 gave the highest degree of accuracy.

Study published by Qureshi etal published in j collPhysiciansSurg Pak 2005Nov;15(11):693-6 showsmortality rate of 28.1% in patients with MPI score more than 26

In study published by Sookmenetalshows every predictor was revealed significant difference between expired and discharged group. It shows increased mortality rates in MPI score more than 30

In our study 100 case of peritonitis was classified into three subgroup that patients with MPI score <21, between 22 and 29 ,>29

Table 8

MPI score	<21	22-29	>29
Mortality	0%	45%	88.9%

The study was found to statistically significant when observed data was subjected to Chi- Square test. Chi- square value was 48.71 and p value was <0.001 .So the results is significant at p value< 0.05

In our study 62 cases were in low risk group with nil mortality, 20 cases in moderate risk group with 45% mortality and 18 patients in high risk group with 89% mortality. Based on this classification we can triage the patients in the government hospitals where the cases are in plenty but the resources are diminished. Most of the mortality was due to multi organ failure due to septicemia occurring in immediate post-operative period. The operative risk is high in these patients and the results are poor. The patients with a MPI score less than 21 usually did well post operatively with minimal complications. But the bulk of morbidity was found in the group having a MPI score between 22 to29 and more than 29. More care may be needed for these patients who with proper care will do well but with a little of neglect can sink towards their deaths.

SUMMARY

Peritonitis is the most common surgical emergency attended in a hospital. Study showed a male predominance. The ratio is approximately 2:1

Most common etiology is acute appendicitis followed by duodenal perforation.. Major cause of post-operative morbidity was wound infection and respiratory complications.

The complications have been most common in the group of patients having a MPI score between 22 and 29, whereas those who have a score above 29 have higher mortality.Mortality was due to multi organ failure

The scores below 21 has got good prognosis and mortality is 0% in this group. Patients with scores between 22 and 29 the mortality was 45%Patients with scores morethan 29 the mortality was 90%.

We can triage the patientsbased on this classification in the government hospitals where the cases are in plenty but the resources are diminished.

Morbidity was foundhigh in the group having a MPI score between 22 to29 and more than 29. More attention may be needed for these patients who with proper care will improve but with a little of neglect can lead to mortality.

CONCLUSION

Mannheim Peritonitis index (MPI) is simple and objective scoring system to predict the final outcome of patients with peritonitis and intra-abdominal sepsis. It appears more practical than other scoring systems.MPI provides an easy and reliable means of risk evaluation and classification for patients with peritoneal inflammation for early intensive management for better outcome of patient.

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ANNEXURE –I

PROFOMA

Name:

Age /Sex:

Duration of illness:

Type of peritonitis:

Investigations:

Complete hemogram

Blood urea

Serum Creatinine

ABG analysis

X-ray abdomen

USG abdomen

Presence of organ failure (which organ system):

Presence of malignancy:

Origin of Peritonitis (site):

Characteristics of exudate:

Presence of any complication:

Mannheim Peritonitis Index score:

Severity:

Hospital stay:

Outcome:

ANNEXE II
MASTER CHART

SI.NO	NAME	AGE	SEX	IP NO.	DURATION	PERITONITIS	ORIGIN	ORGAN FAILURE	MALIGNANCY	EXUDATE	MPI SCORE	SEVERITY	INTENSIVE CARE	WI	WD	RC	IAA	UTI	ECF	HOSPITAL STAY	OUTCOME
1	Riyas	15	M	58106	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	4	D
2	Suryaprakash	15	M	61422	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
3	Velumani	15	F	57989	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D
4	Gopinath	26	M	66631	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	4	D
5	Radhamani	16	F	53698	3	G	ileal perforation	-	-	c	26	m	d	p	-	-	p	p	-	16	D
6	Kiruthika	15	F	63871	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	5	D
7	Kalidas	24	M	6140	2	G	D U Perforation	-	-	c	18	l	-	-	-	-	-	-	-	12	D
8	Chittanthiya	63	M	4726	2	G	D U Perforation	-	-	c	25	m	d	p	-	p	-	-	-	13	D
9	Rubaanraj	17	M	74163	2	L	appendicitis	-	-	-	4	l	-	-	-	-	-	-	-	3	D
10	Karupusamy	40	M	6041	2	G	appendicitis	-	-	-	10	l	-	p	-	-	-	-	-	6	D
11	Rajkumar	55	M	7985	2	G	D U Perforation	-	-	c	25	m	d	-	-	p	-	-	-	9	D
12	Anitha	17	F	63187	2	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D
13	Baskaran	40	M	10732	2	G	gastric perforation	-	-	c	20	l	-	-	-	-	-	-	-	15	D
14	Natraj	60	M	60496	3	G	D U Perforation	P	-	c	32	s	d	p	-	p	-	p	-	3	E
15	Balasubrsmanian	75	M	64873	3	G	gastric perforation	P	p	c	36	s	d	p	p	p	-	p	p	4	E
16	Mandirachalam	34	M	9663	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
17	Subramani	20	M	9205	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	4	D
18	Abdul saleem	34	M	1248	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
19	Suganya	18	F	74231	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
20	Lakshmi	47	F	11611	3	L	appendicitis	-	-	-	9	l	-	p	p	-	-	-	p	8	D
21	Sangeetha	27	F	13514	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D

22	Chinnapathiram	45	M	64589	3	G	colonic perforation	P	p	f	33	s	d	p	-	p	p	-	-	4	E
23	Uma	27	F	17159	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D
24	Suganya	27	F	16734	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D
25	Gokila	19	F	18227	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
26	Balakrishnan	27	M	19750	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
27	Devadas	53	M	32124	1	G	D U Perforation	-	-	c	21	l	-	-	-	-	-	-	-	7	D
28	Radhkrishnan	48	M	24910	3	G	D U Perforation	P	-	c	32	s	d	p	-	p	-	p	-	5	E
29	Senthil kumar	37	M	36989	2	G	D U Perforation	-	-	c	25	m	d	-	-	-	-	p	-	7	D
30	Abdulmajeed	55	M	49615	2	G	colonic perforation	P	p	f	38	s	d	-	-	p	-	-	-	3	E
31	Saravanakumar	34	M	38547	2	G	D U Perforation	-	-	c	25	m	d	p	-	-	-	-	-	9	D
32	Parameshwar	40	M	56577	3	G	D U Perforation	P	-	c	27	m	d	p	-	-	p	-	-	5	E
33	Mariyan	47	M	52787	1	G	D U Perforation	P	-	c	27	m	d	-	-	-	p	-	-	11	D
34	Raju	47	M	52780	1	G	D U Perforation	-	-	-	10	l	-	-	-	-	-	-	-	7	D
35	Chinnan	30	M	60040	3	G	colonic perforation	P	-	f	29	m	-	-	-	-	-	-	-	3	E
36	Reajeswar	45	F	30710	2	G	colonic perforation	P	-	f	34	s	-	-	-	p	-	-	-	3	E
37	Eswari	35	F	21350	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
38	Suseela	35	F	25812	2	L	appendicitis	-	-	-	9	l	-	-	-	-	-	-	-	3	D
39	Rani	40	F	39971	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	4	D
40	Dandapani	55	F	34806	3	G	gastric perforation	P	-	c	32	s	d	-	-	p	-	-	-	2	E
41	Selvi	43	F	52949	2	G	ileal perforation	-	-	c	25	m	d	p	-	p	-	-	-	16	D
42	Indirani	25	F	43210	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
43	Kayarnikka	52	F	62986	3	G	D U Perforation	P	-	c	37	s	d	-	-	-	-	-	-	2	E
44	Devaraj	45	M	61805	3	G	ileal perforation	P	-	c	27	m	d	-	-	-	-	-	-	2	E
45	Ganesan	64	M	68555	2	G	D U Perforation	P	-	c	32	s	d	-	-	-	-	-	-	2	E
46	Maragatham	32	F	61822	2	L	appendicitis	-	-	-	9	l	-	-	-	-	-	-	-	4	D
47	Marimuthu	67	M	71061	2	G	D U Perforation	P	-	c	32	s	d	-	-	-	-	-	-	1	E

48	Manikandan	21	M	58475	1	G	D U Perforation	-	-	-	10	l	-	-	-	p	-	p	-	7	D
49	Marappan	70	M	62579	1	G	D U Perforation	P	-	-	22	m	d	p	-	p	-	-	-	8	D
50	Sivasubramani	42	M	44336	2	G	gastric perforation	P	-	c	27	m	d	-	-	-	-	-	-	2	E
51	Krishnasamy	65	M	45099	3	G	colonic perforation	P	p	f	38	s	d	p	-	p	-	-	-	2	E
52	Marappan	70	M	45678	1	G	D U Perforation	-	-	c	21	l	-	p	-	p	-	p	-	8	D
53	Chinnapan	48	M	8912	2	G	D U Perforation	P	-	c	27	m	d	-	-	-	-	-	-	2	E
54	Solaimuthu	60	M	46805	3	G	D U Perforation	P	-	c	32	s	d	-	-	-	-	-	-	2	E
55	Annadurai	44	M	63408	1	G	D U Perforation	-	-	c	20	l	-	-	-	p	-	p	-	7	D
56	Kalimuthu	60	M	46213	3	G	D U Perforation	P	-	c	32	s	d	-	-	-	-	-	-	1	E
57	Duraikannan	50	M	46932	2	G	colonic perforation	P	p	f	38	s	d	-	-	-	-	-	-	1	E
58	Babu	47	M	47229	3	G	D U Perforation	P	-	c	27	m	d	p	-	p	p	-	-	4	E
59	Yacob	28	M	43929	3	G	D U Perforation	P	-	c	27	m	d	-	-	-	-	-	-	2	E
60	Kamarul islam	26	M	52500	5	G	ileal perforation	P	p	c	31	s	d	p	p	p	-	-	p	4	E
61	Danapathy	20	M	66570	2	G	D U Perforation	-	-	-	14	l	-	-	-	-	-	-	-	7	D
62	Thangavel	60	M	25590	3	G	unknown	P	p	c	32	s	d	-	-	p	-	-	-	2	E
63	Sarveshvar	16	M	70864	2	L	appendicitis	-	-	-	10	l	-	-	-	-	-	-	-	4	D
64	Karthik	15	M	72295	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	4	D
65	Akash	15	M	78333	1	L	appendicitis	-	-	-	0	l	-	p	-	-	-	-	-	3	D
66	Priya	18	F	53400	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
67	Mahaliyan	20	F	54678	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
68	Manna	36	M	1997	2	G	D U Perforation	-	-	c	20	l	-	-	-	-	-	-	-	7	D
69	Prabhu	15	M	76494	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
70	Mahesh	21	M	4563	1	G	D U Perforation	-	-	c	16	l	-	p	-	-	-	p	-	7	D
71	Periyasamy	29	M	7373	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
72	Channiyan	48	M	8912	2	G	D U Perforation	P	-	c	27	m	d	p	-	p	-	-	-	11	D
73	Jesna	15	F	72360	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D

74	Kalimuthu	79	M	10445	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
75	Shalijesh	24	M	14904	1	G	D U Perforation	-	-	c	16	l	-	-	-	-	-	-	-	8	D
76	Aamsy	25	F	73941	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
77	Poornima	15	F	53893	2	L	appendicitis	-	-	-	16	l	-	p	p	-	-	-	-	5	D
78	Siva	19	M	19621	4	G	D U Perforation	P	-	c	27	m	d	-	-	-	-	-	-	2	E
79	Aisha	26	F	74560	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	2	D
80	Revathy	25	F	497	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	2	D
81	Muthuabhinay	15	F	61653	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	2	D
82	Devaraj	28	M	19648	3	G	appendicitis	-	-	c	21	l	-	p	p	-	-	-	-	6	D
83	Mohd rafi	17	M	19533	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
84	Deepa	17	F	5890	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
85	Mantharachalam	50	M	21091	2	G	D U Perforation	-	-	c	25	m	d	-	-	-	-	-	-	9	D
86	Manjan	45	M	24238	3	G	ileal perforation	P	-	c	27	m	d	p	-	p	-	-	-	16	D
87	Saleem	71	M	27242	3	G	gastric perforation	P	p	c	37	s	d	p	-	p	-	-	p	4	E
88	Kammalaya	35	M	28794	1	G	D U Perforation	-	-	-	10	l	-	-	-	-	-	-	-	8	D
89	Murugan	45	M	30250	1	G	D U Perforation	-	-	-	10	l	-	-	-	-	-	-	-	8	D
90	Jothimani	34	F	22663	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
91	Daradarani	16	F	30252	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
92	Kalyani	80	F	31243	2	G	gastric perforation	P	-	-	31	s	d	-	-	p	-	-	p	14	D
93	Ramesh	24	M	30225	1	L	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
94	Parthiban	15	M	36764	2	G	appendicitis	-	-	c	20	l	-	p	-	-	-	-	-	6	D
95	Thangaver	50	M	36909	2	G	ileal perforation	-	-	c	25	m	d	p	-	p	-	p	-	15	D
96	Krishna	48	M	43652	1	G	D U Perforation	-	-	-	10	l	-	-	-	.	-	-	-	7	D
97	Saravanantham	48	M	47571	1	G	D U Perforation	-	-	-	10	l	-	-	-	-	-	-	-	8	D
98	Shahulahmed	15	M	47658	1	G	appendicitis	-	-	-	0	l	-	-	-	-	-	-	-	3	D
99	Shilpa martin	22	F	62208	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D
100	Buvaneswari	17	F	52992	1	L	appendicitis	-	-	-	5	l	-	-	-	-	-	-	-	3	D

KEY TO MASTER CHART

1. SL.NO : Serial number
2. Age : in years
3. Sex : M – male , F- female
4. IP NO: Inpatient number
5. Duration : In days
6. Peritonitis : L – Local peritonitis , General peritonitis
7. Organ failure : p – present, - is no organ failure
8. Malignancy : p - present, - is no malignancy
9. Exudate : c- cloudy , f- fecal
10. MPI score : Mannheim Peritonitis Index score
11. Severity : l – mild , m – moderate , s – severe
12. Intensive care : d – Done , - is not done
13. WI : wound infection , p – present , - is not present
14. WD : Wound dehiscence ,p – present , - is not present
15. RC : Respiratory Complications ,p – present , - is not present
16. IAA : Intra-abdominal abscess ,p – present , - is not present
17. UTI : Urinary tract infection ,p – present , - is not present

18. ECF : Enterocutaneous fistula ,p – present , - is not present

19. Hospital stay : in days

20. Outcome : D – Discharged , E - Expired